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EPISTAXIS — A REVIEW AND MANAGEMENT OF 63 PATIENTS ADMITTED TO THE EAR, NOSE AND THROAT WARDS OF THE GROOTE SCHUUR HOSPITAL DURING 1960

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INTRODUCTION

Epistaxis is a symptom, not a disease. Although no latent lesion is detected in most instances, a careful, thorough investigation in every patient presenting with epistaxis should be made.

Most cases of epistaxis are easily dealt with, either by the general practitioner or by a specialist consultant. Only a minority of patients with epistaxis require hospital admission. In these, bleeding is profuse and uncontrollable, and the patients may be shocked or may become shocked.

The primary aim of treatment is to arrest bleeding, to replace blood loss with blood (if the bleeding is at all severe), and to look for and treat an underlying cause.

During 1960, 80 patients whose presenting symptom was epistaxis, were admitted to the ENT wards of Groote Schuur Hospital. The records of 17 were not available and these have been excluded from certain sections of the review. Table I shows the number of admissions compared with the number admitted for tonsillectomy and/or adenoidectomy. It will be seen that 3·8% of all admissions were for epistaxis.

TABLE I. ENT ADMISSIONS TO GROOTE SCHUUR HOSPITAL DURING 1960

	Whites	*Non-Whites*	*Total*	%
Total number of patients admitted	1,021	1,070	2,091	100·0
Number admitted for tonsillectomy and/or adenoidectomy	436	315	751	35·7
Number admitted for epistaxis	30	50	80	3·8

SITES OF EPISTAXIS

In the vast majority of patients, bleeding occurs from Kisselbach's plexus, in the lower part of the nasal septum (termed Little's area). Bleeding here is readily visible with a good light and nasal speculum, and is easily controlled.

If no bleeding is visible on inspection, it means that blood loss is coming from far back, and in all probability from an inaccessible site. Roberts[1] described this site of bleeding as being on the posterior part of the inferior turbinate, and also described a method of dealing with this. It is, however, not an easy procedure and one that can only be carried out with the patient anaesthetized.

AETIOLOGY

The presence of epistaxis calls for an investigation into its aetiology, as thorough as if it were haematuria or irregular menstrual bleeding.

At all ages the commonest cause of epistaxis is a spontaneous bleed, i.e. no obvious underlying cause is to be found.

Physiological Causes

These are relatively uncommon and always temporary. They include:

1. Violent exertion — violent sneezing or coughing.
2. Extremes of heat or cold.
3. Congestion at the menstrual period (vicarious menstruation).
4. Rarefaction of the air, as in mountaineering, or flying in aeroplanes or space-craft.

Pathological Causes

1. *Local:*
   - (*a*) Trauma — accidents (blow on nose, fractured skull), operation, foreign body, or 'nose picking'.
   - (*b*) Inflammatory — acute (diphtheria), chronic (atrophic rhinitis, syphilis, tuberculosis, or leprosy).
   - (*c*) Neoplastic — benign (polyp, or nasopharyngeal fibro-angioma), malignant (carcinoma).
   - (*d*) Vascular — *spontaneous*, or hereditary haemorrhagic telangiectasia.
   - (*e*) Allergy — allergic rhinitis (in children).
2. *General:*
   - (*a*) Infective fevers — influenza, typhoid fever, glandular fever, or rheumatic fever.
   - (*b*) Hepatic disease — caused by portal hypertension or lowered prothrombin.
   - (*c*) Pulmonary disease — chronic bronchitis and emphysema.
   - (*d*) Cardiac, vascular, or renal — mitral stenosis, hypertension, or nephritis (acute and chronic).
   - (*e*) Blood diseases — purpura, polycythaemia, leukaemia, or scurvy.
   - (*f*) Drugs — salicylates, quinine, or anticoagulants.
   - (*g*) Psychological.

Table II gives the age, sex and race distribution of 80 patients admitted to hospital for epistaxis. It will be noted that in Whites epistaxis is rarely troublesome enough before middle-age to require admission, and that the peak incidence is in the 61 - 70-year age group. In non-Whites, on the other hand, many more younger patients were admitted and the peak incidence is in the 41 - 50-year age group.

Although the series is a small one, Table II shows that the sex incidence in Whites is approximately equal, where-

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TABLE II. AGE AND SEX DISTRIBUTION OF ADMISSIONS FOR EPISTAXIS

Age in years	Whites			Non-Whites		
	Male	Female	Total	Male	Female	Total
0 - 10 ..	—	—	—	2	1	3
11 - 20 ..	—	—	—	1	—	1
21 - 30 ..	—	—	—	8	3	11
31 - 40 ..	1	—	1	5	2	7
41 - 50 ..	3	1	4	2	10	12
51 - 60 ..	4	4	8	—	6	6
61 - 70 ..	5	5	10	3	1	4
71 - 80 ..	2	3	5	1	5	6
81 + ..	—	2	2	—	—	—
Total ..	15	15	30	22	28	50

as in the non-White group there are more females than males.

Table III shows the relative incidence of causes in the 63 patients whose records were complete. It will be seen that hypertension and unknown aetiology constitute about 75% of the causes. In the traumatic group, 3 followed

TABLE III. CAUSES OF EPISTAXIS IN 63 CASES

Causes of epistaxis	Whites		Non-Whites		
	Male	Female	Male	Female	Total
Unknown	5	3	11	11	30
Hypertension ..	3	5	—	9	17
Traumatic	1	—	4	1	6
Coryza	1	—	1	—	2
Tuberculosis ..	—	—	2	—	2
Dilated vessels ..	—	—	1	1	2
Congenital telangiectasia	1	—	—	—	1
Carcinoma of antrum	—	—	1	—	1
Carcinoma of liver ..	—	—	1	—	1
Pregnancy	—	—	—	1	1

operation and 3 followed a blow on the nose. In 1 patient, epistaxis was related to pregnancy, the mechanism being similar to that producing varicose veins.

MANAGEMENT OF EPISTAXIS IN HOSPITAL

Patients admitted to hospital for epistaxis have usually bled in the past, or are bleeding profusely at the time of admission, and are therefore tense and anxious. A calm and purposeful attitude on the part of the attending practitioner is thus indicated. The patient should be told that the epistaxis will cease with treatment.

Bed Rest and Sedation

On admission, the patient should be confined to bed with no toilet privileges. At least 24 hours should elapse after cessation of bleeding before the patient is allowed out of bed. Most patients find difficulty in using a bedpan and, since straining at stool is likely to start further bleeding, purgatives should be given to keep the motions soft.

Morphine, gr. ¼, should be given on admission as a sedative if none was administered in the preceding 4 hours. This may be repeated if necessary. 'Phenergan', 50 mg., is an excellent sedative for an average adult and should be given 2 - 3 times in the 24 hours after admission, combined with phenobarbitone, grs. 1 *t.d.s.* The phenobarbitone is continued for 4 - 5 days. Sedation is an important factor in the management of epistaxis.

Assessment of Blood Loss

The patient and his relatives are usually poor witnesses of the amount of blood lost. Clinical assessment is far more important, though of necessity it is also only a rough guide. The pulse rate and blood pressure should be noted and recorded every half-hour. The haemoglobin should be estimated on admission and repeated daily (or more often if necessary). Blood loss through the anterior nares is usually apparent, but the posterior pharyngeal wall should always be inspected for active bleeding.

No time should be lost, if the condition warrants it, to obtain compatible and, if possible, fresh blood for transfusion.

Dealing with the Bleed

All that is required in most patients is to arrest the haemorrhage in the manner described below. In some instances, however, blood loss may be so severe that replacement is necessary. In these, an assistant should deal with the blood transfusion.

A cotton-wool pledget, soaked in 1 drachm of cocaine (20%) and adrenaline (1: 1,000), is placed in each nostril and kept *in situ* for 10 - 15 minutes. At the end of this time both pledgets are removed and bleeding points are looked for with a nasal speculum and the aid of a strong light.

In most cases, no active bleeding is seen, either because the bleeding is from far back, or because of the presence of blood clot. The patient should be encouraged to blow his nose gently to dislodge the clots.

If bleeding points are seen, they should be painted with silver nitrate or cauterized with an electric cautery needle. (Eight patients in the series, or 12·7%, were cauterized.)

If no bleeding point is seen, the anterior part of the nose is packed under direct vision with ribbon gauze soaked in BIPP, using Tilley's forceps. A common mistake in practice is to pack the anterior nares for only about 1 - 2 inches. Such packing is quite inadequate, since it fails to arrest haemorrhage, and may result in blood trickling down the nasopharynx without being suspected. In the majority of cases adequate anterior packs control the bleeding. They are left in position for 36 hours and should then be removed.

It is wise to pack *both* nostrils, since this provides additional firm pressure and discourages the patient from blowing his nose.

In a minority of cases — 7 (11%) of the series — anterior plugs failed to control the bleeding. In such a case it is necessary to insert posterior nasal packs. For this purpose tonsillar swabs are frequently used. A rubber catheter is passed through the nostril and the end is extracted through the mouth. The pack is tied to this end and pulled firmly to fit in the posterior nasal space. The two ends of the string on the swab are fixed to the cheek by means of 'elastoplast' strapping. Anterior packs are then inserted as before.

If bleeding still persists, the postnasal space may be packed with fibrin foam ('oxycel') or a pint of fresh blood may be given. In very rare cases the patient may have to be taken to the theatre and anaesthetized, and the nose packed as described by Roberts.[1]

Clotting Agents

Non-specific substances like 'adrenosem' (which is known to produce irreversible psychosis) and 'haemoklot' are mentioned only to be discarded. Vitamin K and its analogues have a limited rôle if liver disease is present. Ergometrine, 0·5 mg., or, more recently, 'premarin', 20 mg. intravenously (or 5 mg. *q.i.d.* for 4 - 5 days), have been used. In the series premarin was used in 14 cases (22·2%). The efficacy of oestrogen compounds in controlling nose bleeds is not proved, but their use in intractable cases is unquestionably worth a trial.

Reassurance of the Patient

Reassurance should be an active adjuvant used by the attending physician in the treatment of epistaxis. All patients with nose bleeds should be told that the bleeding will stop in time, with treatment. In most instances the patients are distressed and nervous. Their relatives and private practitioner are often in a similar state of mind. A calm, efficient and industrious bearing on the part of the hospital staff goes a long way towards winning the patient's confidence. It helps, too, to control the bleeding.

It is equally important that the patient should be comfortable in bed. The face, pyjamas, and bed-clothes should be kept free of blood by the nursing staff. A kidney-dish should be available, in which all blood is collected and measured. The patient should be propped up in bed so that all blood can be collected and not allowed to trickle down the nasopharynx. It is customary to apply an ice-pack to the forehead; this should be used only if the patient feels comfortable with it.

Antibiotics

A combination of penicillin and streptomycin in the usual dosage is given to prevent secondary infection. This is especially important in the case of posterior nasal packs which are retained for longer than 24 hours.

Hospitalization and Transfusion

Table IV shows the number of days that patients with epistaxis were hospitalized. Of the 63 patients, 23 required blood transfusion, i.e. 36·7%. The average was 6 pints per patient, but since one patient alone required 45 pints, the true average is about 4 pints. One elderly patient in this series died because of incorrect assessment of blood loss. Five patients were re-admitted with further epistaxis.

TABLE IV. DAYS SPENT IN HOSPITAL BY PATIENTS ADMITTED FOR EPISTAXIS

Days	*Whites*			*Non-Whites*		
	Male	*Female*	*Total*	*Male*	*Female*	*Total*
1	—	—	—	1	—	1
2	1	1	2	4	3	7
3	1	2	3	3	9	12
4	2	4	6	1	3	4
5	3	2	5	3	1	4
6	2	2	4	4	4	8
7	1	1	2	2	2	4
8 - 14	4	2	6	2	3	5
15 - 21	—	1	1	2	2	4
22 - 28	1	—	1	—	1	1

INVESTIGATIONS TO ESTABLISH A CAUSE

A cause for the epistaxis should always be looked for (though in the majority of cases this is not found). A careful history and examination of the patient are imperative. Certain special investigations may have to be done.

The following scheme (though not complete) is given as a guide to determine a cause for epistaxis:

History: (*i*) Trauma or foreign body; (*ii*) bleeding tendency; (*iii*) drugs, e.g. anticoagulants or salicylates; (*iv*) fever or known contact with exanthemata; and (*v*) loss of weight.

Examination: (*i*) Weight loss; (*ii*) skin eruption; (*iii*) jaundice, anaemia; (*iv*) enlarged liver or spleen; and (*v*) blood pressure.

Special investigations: (*i*) Fundoscopic examination; (*ii*) X-ray chest; (*iii*) blood investigation — (*a*) smear and white-cell count, (*b*) ESR, (*c*) liver-function tests, (*d*) blood urea, (*e*) Wassermann reaction; and (*iv*) urine.

If a cause is established, this will be treated in its own right.

SUMMARY

The symptom of epistaxis is discussed, highlighting the types of nasal bleeding encountered, their causation and management.

During 1960, 80 patients with epistaxis were admitted to the ENT wards of Groote Schuur Hospital. It was found that the sex incidence of admissions was more or less equal, but that the non-Whites admitted were about 20 years younger than the White group. In younger patients the males predominated, but in the elderly the females were in the majority.

A plea is made for all intractable bleeds to be referred to hospital before blood loss is severe enough to require transfusion.

It is emphasized that all cases of epistaxis warrant full investigation to exclude underlying local lesions or general diseases. Only if such investigation proves negative, can the epistaxis be labelled spontaneous, idiopathic or primary.

My thanks are due to Dr. D. J. Roux, Head of the ENT Department, Groote Schuur Hospital, and to Drs. G. Kuschke and J. Levit (Registrars).

To Dr. J. G. Burger, Medical Superintendent, Groote Schuur Hospital, thanks are due for permission to quote the figures.

REFERENCE

1. Roberts, J. (1958): Practitioner, **180**, 211.

A FURTHER NOTE ON COSTING

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In an earlier communication on the costing of an anaesthetic service,[1] the average cost of administering an anaesthetic at Groote Schuur Hospital during 1955 was reported, and the figure was broken down into items, which included salaries, drugs, anaesthetic gases, and equipment, in order to show what contribution each item made to the overall cost. The present

report notes the costs of the same service in 1960 and draws attention to several interesting developments in the theory and practice of costing and financing hospital and medical services.

Anaesthetic Costs in 1960

The recording of costs has been continued at Groote Schuur Hospital and in 1960 there was a substantial increase on the figures for 1955. These figures are set out in Table I, with the figures from 1955 for comparison. Several points should be borne in mind in scrutinizing the figures. The most important is the fact that the number and nature of the medical staff

TABLE I. COMPARISON OF ANAESTHETIC COSTS AT GROOTE SCHUUR HOSPITAL IN 1955 AND 1960

	1955	*1960*	*Per cent increase or decrease*
Costs:	£	£	
Salaries	19,170	29,634	+ 55
Gases	8,344	10,103	+ 21
Drugs	2,038	3,099	+ 52
Equipment	1,284	2,255	+ 75
Total	30,836	45,091	+ 46
Number of anaesthetics ..	16,151	16,422	+ 1·6
Staff:			
Consultants			
Full-time	3	8	+ 166
Part-time	5	9	+ 80
Trainees	12	8	− 33
Unit cost per anaesthetic ..	£1.18.2 (R3.82)	£2.15.0 (R5.50)	+ 46

of the department altered appreciably. This is reflected in the table and it will be noted that, in addition to an increase in the total number of staff, there was a quite substantial increase in the number of consultant (specialist) anaesthetists and a *pari passu* decrease in the number of registrars.

Two factors contribute to this alteration in policy, one being the decline in the number of people offering themselves for specialist training, and the other being the alteration in the pattern of surgical care, especially the development of cardiac and vascular surgery, which has demanded a much higher calibre of anaesthetic service. This alteration in pattern has a further effect upon the unit cost of an anaesthetic, because these operations are time-consuming, so that in 1960 there was only a small increase in the number of operations performed as compared with the number for 1955, despite the increase in the number and ability of the anaesthetists. In the intervening years there has also been a further upward adjustment of salary scales, while the cost of drugs, gases, and equipment has increased, in addition to an increase in the quantities used.

Although the unit cost of an anaesthetic rose from the figure of £1.18.2 (R3.82) in 1955 to £2.15.0 (R5.50) in 1960, the internal ratio between itemized costs remained approximately the same. This is to be expected in the light of the work of Seale, which is discussed later.

Other Investigations

Only one similar investigation has been brought to our notice since 1955. In 1960 Shackleton[2] estimated the total expenditure and the unit cost per patient in one of the general hospitals in the Southampton group of hospitals. He found the total expenditure to be £22,945 (R45,890) and the unit cost £3.13.9 (R7.38). In one of the cottage hospitals of the group the unit cost was £5.17.2 (R11.72), while the unit cost for the group as a whole was £2.13.8 (R5.37). It will be seen that our costs compare very favourably with those in Great Britain.

While there are no other comparable figures available, Seale[3-5] has published a series of papers analysing the theories underlying the cost of providing medical and health services of various kinds. The International Labour Organization has also published a Report on the Cost of Medical Care.[6]

It is apparent from these studies that the expenditure on medical care, irrespective of whether it is wholly or partially within the framework of a national health service, or is completely or almost completely on a fee-for-service basis, varies between 3% and 5% of the gross national income. The proportion will vary within these limits, tending to be greater during years of depression and smaller during years of prosperity. A principal reason for this is the slow rate of change in expenditure on salaries and wages which constitute the major drain on available funds. For example, it takes a minimum of 6 years to train a doctor to the point where he can undertake productive work in a medical service, so that if it is necessary to double the number of doctors in such a service this cannot possibly be achieved in less than 6 years. Since facilities for training the increased number must also be provided, it will probably be at least 8 - 10 years before any concrete result of the decision to increase the number of doctors will be evident.

Fixed and Variable Costs

Since the expenditure on wages and salaries does not vary appreciably from year to year, it falls within the category of fixed costs. This category also includes depreciation on capital assets, such as hospital buildings, and the cost of providing heat and light. Whether a hospital be full or half empty; whether the medical and nursing staff be overworked by an epidemic or wasting time because there is too little illness to keep them occupied; these expenditures continue at almost the same level and amount to 70 - 80% of the total spent on providing health service or medical service. The balance is spent on such things as drugs, gases, equipment and food and, since the use of these items will depend directly upon the number of people using the service, the costs will vary, sometimes from day to day. Almost half of these variable costs is devoted to drugs, but this constitutes only 10% of the total cost of the service, so that the total cost cannot be reduced by more than 10% even if all drugs were outlawed.

It is interesting to note from the report of the International Labour Office that, contrary to general belief, while expenditure on medical care is in general rising, it is rising more slowly and to a lesser degree than is the average national income. Hence, the proportion of the national income expended on medical care is static or may even be dropping.

Arising from the observation that drugs account for about 10% of the cost of a medical service, it should be noted that in providing an anaesthetic service this item occupies a much greater proportion of the total cost of the service (29% in 1960). Thus, economy in the use of anaesthetic agents can materially alter the cost of the service. In fact, when, in 1959, the use of halothane was introduced into the service at Groote Schuur Hospital, it was found that this caused an increase of 30% in the overall cost of each anaesthetic in which this agent was used, making the expenditure on drugs and gases a half of the total expenditure on the anaesthetic itself.

Today the cost accountant is the most important person in industry and commerce. He is the architect of a sound economy, and the medical profession would do well to learn to understand his way of thinking and the language he uses. It is only by so doing that we shall be able to avoid a nationalized health service, or that we shall be able to ensure that we enter such a service on an equitable basis.

SUMMARY

The overall and unit costs of providing an anaesthetic service in 1960 are compared with those for 1955.

Recent developments in the theory of costing medical care are noted and discussed.

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4. *Idem* (1960): *Ibid.*, **1**, 1399.
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6. *The Cost of Medical Care* (1958): Geneva, Switzerland: International Labour Office.

EDITORIAL : VAN DIE REDAKSIE

ENDOTRACHEAL ANAESTHESIA: OBSCURUM PER OBSCURIUS

Some months ago the lay press reported the sudden death of a well-known personality which was caused by an abnormal communication between the nose and the extradural space. It appears that the anaesthetist inflated his apnoeic patient before laryngeal intubation, and death occurred almost immediately from herniation of the brain. More recently an established teacher of anaesthesia reported a number of iatrogenic catastrophies and concluded, rightly or wrongly, that something is amiss in modern anaesthetic practice.[1]

Anaesthetist-apologists responded with great vigour, and this correspondence was welcomed because it was felt that in encouraging dignified polemics on a scientific level, which might have led us *not* 'to come out by the same door as in (we) went', a service was being rendered to the profession. However, the tone of some of the correspondence was not altogether satisfactory in that the degree of dispassionate enquiry that was expected had not been achieved. To be sure, revealing and valid arguments were used by some correspondents; these contributed towards achieving the aim of objective discussion of an important and controversial subject. But examples of *argumentum ad hominem*, in which blows were directed against the *man* instead of his *argument*, also abounded, and this reflects the kind of approach to an academic and clinical problem which we can ill afford to borrow from some of our less discerning politician-friends.

Experienced practitioners in South Africa often claim that their patients for caesarean section, for example, did better with 'vinethine' and ether on an open mask than nowadays with pentothal and muscle relaxants. It must be remembered that in our country most anaesthetics are given, of necessity, not by the specialist, but by the general practitioner — as Dr. Jones[2] himself pointed out in this *Journal*. This is by no means to be construed as a particularly evil thing. France, for example, is in this respect much worse off than we are, but it does mean that the modern techniques described in the specialist journals need to be adapted to local conditions. Thus, laryngeal intubation is clearly contraindicated if facilities are not available for inflation of the lungs with oxygen, or where the anaesthetist has not been instructed in the technique. As a corollary, it is equally imperative to desist from the use of muscle relaxants if the technique of laryngeal intubation has not been thoroughly mastered.

Technically there are three manoeuvres an anaesthetist must master completely before he can administer a safe anaesthetic: the intravenous, endotracheal, and 'spinal' techniques. Provided he takes some interest in the administration of anaesthetics to surgical patients, especially during his compulsory year in hospital practice, there is no reason why the general practitioner need have any fear of practising anaesthesia, particularly in the rural areas of South Africa; often enough he has no option anyway. But he will do well to heed the advice of people like Dr. Jones, who have made a special study of the frequently invidious position of the general-practitioner-anaesthetist. For his part it behoves the specialist to consider the needs of our population in the light of *available* anaesthetists, rather than deploring the slightest deviation from the high present-day specialist standards in clearly sincere attempts to cope realistically with the great shortage of specialist anaesthetists — a problem that is certainly not going to be solved in the near future. Nowhere is the precept to do no harm more important than in anaesthesia; and while this is usually 'terribly simple', it can so easily (at least occasionally) become 'simply terrible'.

Any attempt to mitigate against such iatrogenic disaster clearly merits the serious attention of all concerned. And if the lay press abuses its access to such writings, let us remind ourselves that we, too, have a duty to report cases like those mentioned in the first paragraph of this article, rather than to leave to laymen the reporting of cases. This can only help to dispel obscurity which is in danger of being made more obscure by controversy and negative criticism.

1. Jones, C. S. (1961): S. Afr. Med. J., **35**, 421.
2. *Idem* (1959): *Ibid.*, **33**, 1036.

VAGINALE ONDERSOEK TEN TYDE VAN SWANGERSKAPSBESPREKING

Dit lyk of daar 'n mate van aarseling of traagheid by geneeshere is om 'n swanger vrou vaginaal te ondersoek by die eerste — en dus die beste — geleentheid, nl. die heel eerste swangerskapsbesoek. Nog veel erger — sommige vrouens word nooit vaginaal ondersoek op enige tydstip voor of selfs met kraam nie. Veral is die eersgenoemde versuim sterk af te keur.

Die meeste vrouens kom bespreek die reëlings, ens. vir hul verlossings waarskynlik eers nadat hulle al drie maande lank swanger is, d.w.s. nadat die gevaarlikste miskraamtyd verby is. Dit is buitendien erg te betwyfel of oordeelkundige en versigtige spekulum- en vaginale ondersoeke ooit miskraam veroorsaak. Indien in 'n besondere geval iets van hierdie aard vermoed mag word, kan die ondersoek uitgestel word tot na die derde maand, maar nie langer nie. Daar is dus by verreweg die grootste gros van pasiënte geen werklike kontraindikasie vir hierdie soort ondersoek nie. En daar is veelvuldige goeie redes waarom so 'n ondersoek onderneem behoort te word, want daar mag tog infektiewe of ander toestande van die vulva,

serviks, vagina, uterus, of adnekse wees; en hoe kan die dokter daarvan weet as hy nie hierdie organe sistematies ondersoek nie?

Die vulva mag tekens toon van irritasie — iets wat dikwels saamhang met 'n moniliase-vaginitis — 'n vry algemene swangerskapstoestand wat ontsettend lastig kan wees vir die pasiënt. Ander velletsels mag ook aanwesig wees, asook tekens van ander vorms van vaginitis of aangebore vaginale siste of bande van embriologiese oorsprong. Die serviks mag gedupliseerd wees, wat seldsaam is, of dit mag 'n erosie toon, wat 'n ontstellende voorkoms mag hê vir diegene wat hulself nie gewoond maak aan die swanger serviks en sy voorkoms nie. Dit is veilig om biopsies te neem in swangerskap, met die regte tegniek; of om Papanicolaou-smere te neem (hoewel die histologiese vertolkings moeilik mag wees). Daar word beraam dat servikale karsinoom 'n voorkoms van ongeveer 1 tot 2 in 4,000 gevalle het met swangerskap. Dit is dus vanselfsprekend dat hoe gouer dit gevind word, hoe beter in alle opsigte. By geleentheid mag daar ook 'n inkompotente os so raakgeloop word, en tydige terapie kan dan ingestel word.

Die uterus mag in die vroeë maande verkeerd lê, nl. in retroversie. Dit is op sigself van geen betekenis nie, maar die uterus behoort op drie maande abdominaal te wees anders moet gewaak word teen inkarserasie met moontlike uretraversperring. Ook mag die orgaan groter of kleiner as die verwagting op sekere datums wees, en gevolglik kan tweeling-swangerskap, mola, of intra-uterine dood vermoed word, of fibroïdes kan gevind word, wat soms later in die swangerskap degenereer en dan groot diagnostiese probleme kan skep.

Ten slotte mag daar gewasse of groeisels van die ovaria wees. Daar moet onthou word dat hierdie soort abnormaliteit 'n besondere neiging het tot steeldraaiing in swangerskap — 'n toestand wat kan presenteer as 'n akute buiktoestand en wat verwante diagnostiese en terapeutiese probleme kan oplewer. 'n Verdagte mola kan bevestig word deur die gepaardgaande teka lutein-siste en daar mag selfs 'n korpus luteum-sis wees wat sal neig om in die vierde maand te kwyn. Dit word algemeen beskou as raadsaam om chirurgie, waar moontlik, uit te stel tot na die derde maand.

Daar bestaan dus goeie en genoegsame redes waarom 'n swanger vrou sover as moontlik grondig en omvattend ondersoek moet word met die eerste of wel besprekingsbesoek. Die ondersoek moet 'n sorgvuldige vaginale en spekulumondersoek insluit. Om vooraf kennis te dra van moontlike oorsake van latere komplikasies beteken tog dat die helfte van die stryd gewonne is as hierdie komplikasies hul verskyning maak.

NEURO-PSYCHIATRIC DISORDERS IN THE BANTU

I. CONVULSIVE DISORDERS — A PILOT STUDY WITH SPECIAL REFERENCE TO GENETIC FACTORS

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This article is the first report of a long-term investigation which is being carried out at Meadowlands Clinic into neuro-psychiatric disorders occurring among the Bantu in this part of the Witwatersrand area.

The question of the comparative spectrum of neuro-psychiatric disease, both as regards relative incidence and variable pattern of symptoms in different ethnic groups, is but part of the current world-wide movement for the study of the epidemiology of disease in general. In defining the field as a whole one of us[1] drew attention to the work already done at Baragwanath Hospital (the parent hospital of the Meadowlands Clinic) by Reef, Lipschitz and Block[2] demonstrating a difference in distribution of neurological disease (including disseminated sclerosis) in the Bantu as compared with Whites. Moreover, our clinical impression suggests that at Baragwanath Hospital there is a high incidence of gross hysterical states comparable to that characteristic of a less sophisticated era in Europe. The rich prospects for research held out by such observations has led to our embarking on our present series of studies.

The main objects of this study, which is our first investigation in this field, are as follows:

1. A longitudinal study of the incidence of convulsive disorders, mental disorder, and mental deficiency with reference to the ethnic variations of these conditions.
2. An investigation into the genetics of these disorders.
3. A study of congenital neuro-psychiatric diseases in relation to the abnormalities of pregnancy.
4. The assessment of aetiological factors such as nutritional status, alcohol consumption, helminth infestation, primitive beliefs, and socio-economic causes, and the integration of the patient in the family and community milieu.

Phase 1 of this investigation is limited to a study of convulsive disorders.

METHODS OF INVESTIGATION

This project was commenced on 1 September 1959, and the preliminary results assessed on 1 March 1961.

Meadowlands Clinic is a polyclinic where all types of illnesses are investigated. The clinical records are filed in a cohort system of age and ethnic groups. All neuro-

psychiatric diseases are indicated by a system of signals which facilitates abstraction of these from the various filing cohorts. The clinic has been established for 3½ years and, from the issue of cards, it appears that there is nearly full coverage of the population who have attended the clinic at one time or another for various complaints.

All the recorded patients and new patients have been examined at the clinic during the period of this survey. If the diagnosis could not be determined the patients have been admitted to Baragwanath Hospital for further investigations. The homes of all patients were visited by a trained Bantu nurse to obtain additional information on family backgrounds.

Environment and Demography

Meadowlands is a Bantu residential area with a population of close on 60,000. There are 8 administrative zones with separate ethnic-group demarcation, viz. Nguni, Sotho, Shangaan, and Venda races.

TABLE I. ETHNIC COMPOSITION OF MEADOWLANDS TOWNSHIP

Nguni	*Sotho*	*Venda/Shangaan*	*Total*
15,300	29,500	11,500	56,300

Intermarriage does occur among the groups, but the Shangaans and Vendas still maintain many of their primitive customs. The women wear leather skirts and metal rings on their feet. In some households food is imported from the Northern Transvaal and corn is ground in wooden mortars. Many of the children are sent back to the tribal areas for long periods, where they are subject to such diseases as malaria and bilharziasis.

We observe a transition stage between a primitive rural life and the complexities of modern civilization. The mental adaptation to these changes has yet to be assessed. The tribal protective authority has broken down to be replaced by European laws and customs with totally different social disciplines. A complex economic environment has replaced the simple economy of the tribe. Rising costs make it necessary for both parents to work. This is evident in the Nguni and Sotho households where lack of parental care tends to produce malnutrition among infants, and delinquency and a high rate of illegitimacy among the adolescent population. There is marked disintegration of the family unit. This is in contrast to the Venda and Shangaan women, who have maintained their tribal outlook and who, for the most part, do not work in the European sphere of influence. They remain culturally primitive while their menfolk have adopted the urban way of life.

An important cause of neuro-psychiatric disorders is the presence of single or combined nutritional/alcohol factors. In these areas, potent alcoholic beverages of bizarre composition are consumed. The long-term ingestion of such drinks, with an inadequate diet of maize, produces physical and psychological pathology which is not seen in European communities.

It will be noted that the general incidence of these convulsive disorders in this area is approximately 1 per 1,000. This figure is lower than the incidence of 4 per 1,000 quoted in a recent survey by the members of the College of General Practitioners in Great Britain.[3] Every effort has been made to trace these patients in Meadowlands. The dramatic and urgent nature of an attack makes attendance for therapy more likely. Nevertheless, one cannot be certain that patients have not short-circuited the clinic service. However, the impression remains that the incidence is low, and until a comprehensive survey is made, the actual figure must remain uncertain. An interesting feature is the very low incidence among the Shangaan and Venda residents. These people are far more primitive in their outlook and way of life than the Sotho and Nguni races. There is no present evidence that among the more primitive tribes the incidence of epilepsy is lower than among more civilized peoples.

TABLE II. ANALYSIS OF CASES

1. *Age/sex distribution of convulsive seizures*

	0 - 9	10 - 19	20 - 29	30 - 39	40 - 49	50 *plus*	*Total*
M.	7	7	4	1	0	2	21
F.	4	10	7	4	3	1	29
	11	17	11	5	3	3	50

2. *Ethnic variations*

	Nguni	*Sotho*	*Venda/Shangaan*	*Total*
Cases	34	13	3	50
Population	15,300	29,500	11,500	56,300
Cases per 10,000 ..	22·2	4·4	2·6	

Clinical Description of Material

The age/sex distribution of our material is depicted in Table II. This follows the general pattern of the age distribution of Meadowlands residents. Of the patients 21 were males and 29 females.

TABLE III. POSSIBLE AETIOLOGICAL FACTORS

Difficult labour, birth trauma ..	7
Head injury	5
Cysticercosis	1
Cardiovascular accident	1

Possible aetiological factors elicited from the history were obtained in 14 patients and these are shown in Table III. The 5 patients with head injuries were all adults and the injuries severe ones.

TABLE IV. TYPE OF ATTACK

Grand mal	41
Focal	8
Petit mal	2

With the aid of interpreters and relatives it proved possible to ascertain the nature of the attack in all patients except one. This information is recorded in Table IV. Twenty-three patients gave a positive history of warning auras before the attacks — a rather high incidence. The nature of the attacks is shown in Table IV. The 8 patients who gave an aura of dizziness found it very difficult to describe the true nature of their experiences. One patient volunteered that his attacks were precipitated by singing, especially hymns, which produced a feeling of elation — 'Doctor, I feel like a king' — and soon afterwards he would have a *Grand mal* attack. Two patients gave rather unusual auras: one felt as if he could not close his eyes and the other felt as if his eyes were full of tears just before an

attack. There were no epigastric auras (Table V). Forty-one patients had *Grand mal* attacks, 8 had focal attacks, and 2 *Petit mal* attacks. One patient had both *Grand mal* and *Petit mal* attacks.

TABLE V. INCIDENCE AND TYPE OF AURA

Dizziness	8
Visual disturbances	4
Tinnitus	2
Feeling of fear	1
Inability to close eyes	1
Crying	1
Eyes full of tears	1
Palpitations	1
Aphasia	1
Confusion	1
Weakness of right hand	1
Feeling of elation—attacks precipitated by singing	1

TABLE VI. INCIDENCE OF ASSOCIATED PSYCHIATRIC FEATURES

Mental defect	12
Psychosis	1
Behaviour disorder	3

Of the 50 patients seen, 12 were regarded as being mentally defective both on history and personal examination. The degree of the defect in all of these was sufficiently severe to justify institutional treatment. One additional child showed severe behaviour problems, and one adult was frankly psychotic. These patients comprise 28% of our total and are recorded in Table VI. This very high percentage is unusual and may be accounted for by the fact that it is for this sort of complaint that the African is most likely to seek the aid of a doctor. These patients are a great burden to their relatives and, since no adequate facilities exist for their care, they have to remain at home and often prevent an adult member of the family from working.

TABLE VII. FREQUENCY OF ATTACKS: GRAND MAL AND FOCAL EPILEPSY

2 or more per week	6
1 per week	14
1 per month	12
1 attack per 3 months	3
Less than 1 a year	4
Bouts of attacks	4

All the patients' records were carefully scrutinized, the frequency of attacks assessed as recorded in Table VII, and the end-results of treatments were assessed. Despite the presence of adequate facilities for the treatment of their disease, not one patient was fully controlled and not one patient attended regularly for treatment as instructed. All patients and relatives were questioned on this aspect and the following facts emerged: Patients attend at the clinic usually soon after they have had an attack and expect to be given medicines which would produce an immediate cure. When the prescribed drugs failed to stop the attacks, or if the attacks recurred when the drugs were finished, some of them lost faith and did not attend again or they began to attend very irregularly afterwards. The idea of continuous suppressive therapy over a period of many years was completely foreign to them. Even after detailed explanation few of them were prepared to accept this form of treatment.

Fourteen of the patients admitted to having consulted witch-doctors. Thirteen were told that they had offended their ancestors and one that he had been bewitched by a neighbour. Suitable treatment was prescribed for all at a cost varying from £1—£5 (R2—R10). No patient benefited from this treatment and no patient returned for a second course.

The attitude of relatives and friends to our patients was ascertained by members of the nursing staff and ourselves. In no case did we find that relatives were ashamed of having epilepsy in their family. They were not afraid of the disease and did not associate it with evil spirits or demons. They regarded epilepsy as they would any other disease with no special stigma attached to it.

Genetic Aspects

The Bantu clinical material at the Meadowlands Clinic holds out the prospect, because of the large family groups in contrast to the smaller family groups of Whites (in Europe and America) upon which our present knowledge is based, of throwing light on the status of genetic factors in epilepsy. The average size of sibship in the 46 families* upon which the present pilot study was conducted is 5·8 with a range of 1—16.

New light on this question is urgently needed because of the radically conflicting evidence of, on the one hand Conrad [4] (Germany) and Lennox and the Gibbses[5,6] (USA) stressing the prominence of the hereditary factor, and on the other, Alström's Scandinavian figures,[7] which reduce the rôle of genetics in this sphere to the barest minimum, a finding which may in a measure correspond with the incursions into the sphere of idiopathic or cryptogenic epilepsy by the neuropathological findings of our neurosurgical age.

What then are the points at issue within the camp of the geneticists in the sphere of epilepsy which require further work in clarification?

The work stressing the importance of the genetic factor comes, as has already been indicated, from two groups. In Conrad's comprehensive pioneer study[4] the expectancy figures in consanguineous groups of cases diagnosed as idiopathic epilepsy were 4·0% for siblings, 4·3% for two-egg twins and 86·0% for one-egg twins. The similarity of the figure for siblings and two-egg twins, categories which may be equated as to hereditary equipment, and the extremely high concordance rate for one-egg twins with identical hereditary equipment as between co-twins, are eloquent and cogent testimony to the validity of the genetic factor. Kallmann, in reflecting on Conrad's work, taken in conjunction with general considerations concerning the ubiquity of the epileptic mechanism in man under certain conditions of release and its prevalence throughout the animal kingdom, is inclined to the view that the genetic mechanism is polygenic. Lennox and the Gibbses,[5] using dysrhythmia in the EEG as their criterion of epilepsy, record the remarkable finding of 100% concordance in one-egg twins and 25% concordance in those of the two-egg variety — the ideal figure for a fully penetrant single dominant gene. Our two groups of workers are thus in full agreement on the importance of

* The discrepancy from the figure of 50 recorded earlier in this article is apparent, not real — being based on the fact that in 4 cases siblings other than the index cases are affected.

the rôle of genetic factors in epilepsy, differing only in the details of the genetic mechanism involved.

Then, in 1950, came the publication of work by Alström,[7] based on a study of epileptic patients admitted during the years 1925 - 1940 to the neurological clinic of the Caroline Institute of the Serefimer Hospital, the only university clinic for neurology in Sweden at that time. Alström remarks that the patients came from all over the country, but that the urban population, especially that from the capital, was over-represented. He claims at the same time that this sample was otherwise probably a more representative one for patients suffering from convulsive disorders than a sample taken from hospitals for the insane or from institutions for epileptics with their selection of mentally affected patients. The investigation of his 897 index cases with their blood relations began in 1945 and ended in 1950.

Salient findings of this study were as follows. In the first place the expectancy figures for parents, $1{\cdot}3 \pm 0{\cdot}27\%$; for siblings, $1{\cdot}5 \pm 0{\cdot}25\%$; and for children $3{\cdot}0 \pm 0{\cdot}93\%$, were not significantly higher than those in the general population. Secondly, families with epilepsy in members other than in the index case are lacking in the majority, i.e. 92%, of cases. Thirdly, among the 16 pairs of twins of this study, two of which pairs were monozygotic, there was not a single case of concordance as to epilepsy. In this connection Conrad[4] discusses a small sample of 4 unselected single-egg Scandinavian twin pairs (the index case, but not the co-twin, suffering from epilepsy in every case) drawing attention to the lack of agreement with Conrad's and Lennox's series,[5] and pinpointing the fact that 'the probability of getting such a sample at random is less than 0·002 if the "degree of manifestation" were to be the same as in Conrad's and Lennox's series'.

Despite Alström's figures quoted above, which reveal no genetic factor in epilepsy, the examination of individual pedigrees in his series discloses, according to Alström's own admission, a genetic factor — in fact a single dominant mechanism — in approximately 1% (11 index cases belonging to 8 families in his sample of 897 index cases and their families). This is the type of genetic mechanism, it will be recollected, that Lennox and the Gibbses postulated as being operative in their series, but present throughout instead of in only 1% of cases. It is, however, impossible to arrive at a final explanation of the discrepant evidence of the two groups of advocates on the available evidence. That is why our present study has been undertaken with a view to clarifying the issue on new material with the peculiarly favourable feature of large family size. Before proceeding to a description of our genetical pilot study, we should point to possible general sources of the discrepancies between the views of Conrad[4] and the Gibbses and Lennox[5,6] on the one hand, and Alström[7] on the other. The following thoughts come to mind:

1. Accepting the possibility that the epilepsies may be divided into two groups, (*a*) genetic, and (*b*) non-genetic, it is readily conceivable that different samples may contain variable loadings of the two varieties, owing to either (*i*) some selective process in the collection of the sample, as might well be brought about by a clinic, in contrast to a mental hospital, constituting the source of the material, or (*ii*) by the more or less plentiful introduction into a specific country or area of the genetic variant of the disease. (Thus, a generally rare genetic condition such as Huntington's chorea or porphyria may, by the chance of introduction into an isolated area, assume disproportionate dimensions.)

2. Alström's work gives the impression of deficient recording of ages of all categories of blood-relationship, resulting in our often having to be content with nett figures which lack universality in the sense of applicability in all samples, and are thus often misleading as compared with expectancy figures.

Let us now turn to the findings of our own study with the pointers it gives for a more comprehensive investigation.

The 46 epileptic patients have an average sibship figure of 5·8 with a range of 1 - 16. Side by side with the advantage for genetic purposes of working with these large family groups, it should be recorded that in this pilot study it has not been possible to ascertain the ages of all the groups of blood relationship investigated, which include siblings, half-siblings, children, nephews and nieces, cousins, parents, uncles and aunts, and grandparents and their siblings. Such ascertainment of age is essential for expectancy studies, and it is proposed to include this in the full investigation which, according to our plans, will follow the current pilot study. This will entail careful investigation by the social worker attached to the proposed team at house-to-house visits, and in view of the cultural limitations of the groups investigated, even in an urban area, we may have to be content with ages assessed as falling within a defined age range rather than the precise age.

Despite this limitation, our preliminary pilot study is adequate to produce evidence along the following two lines:

1. The percentage of families showing one or more members exhibiting epilepsy in addition to the index case, for comparison with Alström's low figure cited above.
2. The types of genetic mechanism suggested in different pedigrees contained in our material.

With regard to the first point, our material shows an incidence of 13 out of 46 families, i.e. a figure of 28·3%* in contrast to Alström's 0·8%. Statistical computation shows this difference to be significant at the 0·1 per cent level. Thus, even at this early stage, our study has afforded evidence on the side of Conrad and the Gibbses and Lennox on the importance of the genetic factor in epilepsy.

Turning to the second point, analysis of the 13 positive pedigrees (of the 46) shows that 3 of these are strongly suggestive of a penetrant single dominant mechanism and 1 of irregular dominance, while the remaining 9 are equally compatible with irregular dominance, or recessiveness, as indicated in Table VIII. A portion at least, therefore, of these results is in line with the thesis of single dominance of Lennox and the Gibbses, appearing also in that of 0·8% of Alström's cases where a genetic mechanism was conceded by him.

As a conclusion to this pilot genetic investigation, it may be said that the positive findings are sufficiently

* This figure is probably conservative, since cases 10 and 39 in Table VIII each had a relative whose psychosis may well have been epileptic in nature.

encouraging to warrant our proceeding to the more comprehensive survey already alluded to.

(At an impressionistic estimate the anticipation that the rate of questionable paternity in our Bantu group would be unduly high was not realized. This point, however, will be one of the special terms of reference of our proposed extended enquiry.)

TABLE VIII. GENETIC FEATURES

	Identification	*No. of sibs.*	*Relatives affected*	*Probable type of genetic mechanism*
1	J.Ma.	4	Negative	
2	L.K.	15	Negative	
3	S.D.	0	Negative. Inadequate history	
4	D.S.	2	Negative	
5	L.M.	2	Negative	
6	J.Q.	3	One of five children Two siblings and mother	Penetrant single dominant
7	P.D.	3	Negative	
8	T.L.	3	Negative	
9	I.M.	4	Negative	
10	M.M.	4	Negative. Paternal uncle mentally disordered	
11	M.N.	4	Negative	
12	A.M.	6	Negative	
13	J.Mo.	6	Maternal great-aunt on her maternal side. Maternal cousin on her paternal side	Irregular dominant
14	N.M.	3	Negative	
15	S.B.	4	Nephew (1 of 2 sons of an elder brother)	Irregular dominant or recessive
16	B.K.	3	Negative	
17	M.F.	7	1 sibling	Recessive or irregular dominant
18	M.P.	4	1 uncle and 1 of 5 paternal siblings	Recessive or irregular dominant
19	M.Z.	3	1 of 3 other male siblings	Recessive or irregular dominant
20	G.D.	7	Negative	
21	Ja.Mo.	7	Negative	
22	V.K.	7	Negative	
23	M.N.	3	Negative	
24	E.M.	2	Negative	
25	F.N.	5	Negative	
26	P.M.	5	Paternal grandmother	Recessive or irregular dominant
27	M.Z.	3	Mother, 1 of 4 sibs. and only female child	Penetrant single dominant
28	C.N.	7	Maternal aunt and 1 of 4 siblings	Recessive or irregular dominant
29	W.N.	5	Negative	
30	M.Nd.	6	Paternal uncle and 1 of 3 siblings	Recessive or irregular dominant
31	Jo.Mo.	4	Negative	
32	J.Mak.	4	Negative	
33	L.T.	9	Negative	
34	M.H.	4	Negative	
35	El.M.	0	Negative	
36	P.T.	8	Negative	
37	Ru.M.	6	Sister and father	Penetrant single dominant
38	S.D.	0	Negative. Poor history	
39	B.M.	0	Negative. Paternal grandfather psychotic	
40	S.J.	8 (1 twin pair)	Negative	
41	M.Ng.	7	Negative	
42	D.G.	4	Negative	
43	G.N.	12	1 sister and another sister with confusional episodes	Recessive or irregular dominant
44	D.M.	4	Negative	
45	Jo.Mo.	4	1 sibling	Recessive or irregular dominant
46	P.M.	6	Negative	

SUMMARY AND CONCLUSIONS

1. In this article a series of 50 Bantu patients suffering from convulsive disorders, examined at the Meadowlands Clinic in the Witwatersrand area between 1 September 1959 and 1 March 1961, is reported.

2. A description of special features of an ethnic-cultural type that have presented themselves to date in the clinical and social aspects of our patients, is given.

3. A tentative attempt at aetiological analysis is made.

4. The large family size of our Bantu patients, as compared with the modal White family of our time, has permitted a preliminary assessment, substantiating the correctness of the view of the schools that attribute significance to genetic factors in a considerable proportion of epileptics.

5. The purely pilot nature of this study is stressed.

We wish to thank Dr. I. Frack, Superintendent of the Baragwanath Hospital, Johannesburg, for his encouragement of this work, and his permission for the use of clinical material.

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‚STRESS'—DIE ALARMREAKSIE EN DIE ALGEMENE AANPASSINGSINDROOM*

'N KORT OORSIG EN WAARDERING VAN SELYE SE OPVATTING OOR DIE BASIESE FISIOLOGIESE AGTERGROND VAN GESONDHEID EN SIEKTE

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In die mediese wêreld, net soos elders, warrel daar ook gedurigdeur ‚winde van verandering'. 'n Paar jaar gelede, byvoorbeeld, het die handelsreisigers 'n florerende besigheid geken deur die verkoop van ‚Ultrasoniese' masjiene—en vir 'n periode is baie pasiënte deur baie geneeshere van baie kwale genees, of in elk geval behandel daarvoor.

Die era van die ‚Kux-isters' is ook nog gedeeltelik met ons—alhoewel ek steeds tevergeefs wag op die veelbeloofde gedokumenteerde kliniese verslae wat sommige van hulle beloof het om te publiseer.

* 'n Bespreking gehou by geleentheid van 'n opknappingskursus by die Nasionale-hospitaal, Bloemfontein, in September 1960.

Sonder om selfs by implikasie te insinueer dat daar enige regverdige vergelyking getref kan word tussen bogenoemde twee voorbeelde van geleentheidsterapie en die volgende, kan dit gesê word dat ons op die huidige oomblik weer verkeer in die era van die kortisoon-preparate. Kyk maar na die lang lys van aansprake wat daar vir hierdie preparate gemaak word.

Onder die omstandighede het ek noodgedwonge gevoel om my eie kennis op te knap oor hierdie hormoon se gebruik, en dit het gelei tot verwysings na die sogenaamde hipofise-bynier-as, en dit weer na Selye se ‚Stress'-sindroom — wat alles vir my so interessant was dat ek gewaag het om te dink dit mag 'n gepaste onderwerp wees vir hierdie bespreking.

Definisies van ‚Stress'

‚Stress' is 'n onbevredigende woord — vatbaar vir meer as een interpretasie. Selye self het gevind dat hy dit nie kon vertaal as hy in ander tale as Engels oor sy onderwerp besprekings hou nie. Een ding waarmee dit nie verwar moet word nie, is die idee van geestesspanning of ‚mental stress'. Geestesspanning kan ‚stress' aanbring, maar is nie ‚stress' in Selye se sin van die woord nie.

Selye gee dan ook, in sy poging om die begrip te verduidelik, meer as een definisie, en die definisies stem nie almal in alle opsigte ooreen nie. Byvoorbeeld, op een plek omskrywe hy dit baie vaag as ‚the ordinary wear and tear of life'.

Dan verduidelik hy weer: ‚Stress designates the sum of all changes of a non-specific character, effected in the body by any factors which act upon it.' Soms weer raak hy byna digterlik wanneer hy liries uitroep:

‚The soldier who sustains wounds in battle, the mother who worries about her soldier-son, the gambler who watches the races, the horse and jockey he bets on; — they all are under stress.'

‚The beggar who suffers from hunger and the glutton who overeats; the little shopkeeper with his constant fears of bankruptcy and the rich merchant struggling for yet another million; — they also are under stress.'

‚The housewife who tries to keep her children out of trouble, the child who scalds himself, and especially the particular cells of the skin over which he spilled the boiling coffee; — they too are under stress.'

‚What then', vra hy self, ‚is this mysterious condition which the most different kinds of people have in common with animals, and even with individual cells — at times, when much of anything happens to them?'

Dit dan, hierdie begrip ‚stress' soos geïnterpreteer deur Selye, is wat ek nou wil bespreek.

Omvang van die Onderwerp

Omdat daar so veel geskrywe is oor hierdie saak, is dit onmoontlik vir 'n enkele bespreking daarvan om meer as 'n oorsig en inleiding te wees. Om dit enigsins ordentlik te bespreek sou op die minste 'n hele reeks lesings oor die verskillende fasette daarvan vereis.

Sedert Hans Selye in 1936 sy eerste beskeie artikel in die tydskrif *Nature* die lig laat sien het onder die titel: *A syndrome produced by various nocuous agents,* het sy opvatting oor die belang en omvang daarvan geweldig uitgebrei om 'n al hoe wyer veld te dek. Waar hy destyds 'n onbekende jong navorser in 'n klein laboratorium sonder faam of veel fasiliteite was, is daar sedertdien al (teen ongeveer 1956) meer as 25.000 artikels deur hom en andere aan die probleem gewy, en het hy self oor die hele beskaafde wêreld in baie tale lesings daaroor gelewer. Hy beklee vandag die pos van hoof van 'n moderne en uitgebreide Instituut van Eksperimentele Medisyne en Chirurgie, verbonde aan die Universiteit van Montreal in Kanada.

In enige uiteensetting van sy werk wat onderneem word, is dit nodig om te onderskei tussen sy werklike eksperimentele bevindinge en die natuurwette daardeur gedemonstreer, en sekere gevolgtrekkinge en teorië wat hy op die basis van sy bevindinge as moontlikhede voorstel.

Die Belang van Selye se Bydrae

Of Selye se bydrae tot ons kennis wel van enige permanente waarde gaan wees, sal u vir uself oor moet besluit. Die Britse chirurg en skrywer, Ogilvie, het beweer: ‚Dr. Selye's contribution is perhaps the greatest contribution to scientific medicine in the present century'. Selye self, wat andersins beskeie voorkom, is van mening dat: ‚The significance of this kind of research is not limited to fighting this or that disease. It has a bearing upon all disease, and indeed upon all human activities, because it furnishes knowledge about the essence of life. To understand the mechanisms of stress gives physicians a new approach to the treatment of illness, but it can also give us all a new way of life, and a new philosophy to guide our actions, in conformity with natural laws.'

As dit waar is, soos iemand eendag gesê het, dat, om bevredigend te wees, ‚The practice of medicine should be both a *way of living,* no less than a *means of living',* dan is dit die moeite werd om te hoor of Selye enigsins sy eie onderneming om ons die weg te wys na ‚a new way of life', nakom.

'n Opsomming van Selye se Idee

Die hoofbydrae van Selye se teorieë kan hierin gevind word dat, waar ons hoofsaaklik opgelei is om te soek na spesifieke etiologiese faktore wat 'n spesifieke uitwerking het, met spesifieke patogenetiese gevolge wat spesifieke simptome en tekens veroorsaak en spesifieke terapie vereis, Selye, in teenstelling hiermee, daarop nadruk lê dat daar altyd, onderliggend tot en dikwels versluier deur spesifieke veranderinge in die liggaam gedurende siekte sowel as gesondheid, 'n *non-spesifieke* element van verandering en reaksie herkenbaar is.

Dis hierdie element wat hy aandui met die woord ‚stress': ‚Which designates the sum of all the non-specific factors (including normal activities such as muscular work, worry, rage, etc., as well as disease-producing organisms, drugs, injury, cold, starvation, heat, etc.) which can act upon and stimulate the body.'

Hy erken dat hierdie begrip van ‚stress' eintlik 'n *abstrakte* idee aandui, iets wat nie as 'n onafhanklike entiteit bestaan nie, maar in die liggaam, gedurende die lewe, saam voorkom met enige spesifieke veranderinge en reaksies wat deur prikkels voortgeroep kan word, en eers as sodanig herkenbaar is nadat, van die gesamentlike veranderinge, die spesifieke veranderinge deur ons herken en afgetrek is.

Verder beweer hy dat hierdie abstrakte toestand van ‚stress', homself toon (‚manifests itself') en dan as sodanig herkenbaar word, omdat dit funksioneer deur middel van 'n meganisme wat saamgestel is uit 'n gekoördineerde tussenwerking van sekere endokrienkliere in die liggaam.

Dit is hierdie morfologiese veranderinge in die betrokke kliere, plus die waarneembare hormone wat deur hul afgeskei word, wat meetbaar is en dit dus moontlik maak vir die abstrakte toestand om nie alleen herken te word nie, maar eksperimenteel demonstreerbaar en analiseerbaar te wees.

Ten slotte vind hy ook dat hierdie non-spesifieke fisiologiese reaksie tot stimulus die liggaam agterna laat met 'n verhoogde aanpassing en weerstand teen verdere skade wat mag ontstaan as gevolg van toekomstige blootstelling aan stimulus of irritasie.

Wat bedoel hy nou eintlik met al hierdie bewerings? Hoe het hy daartoe gekom om daaraan te dink? Hoe demonstreer en bewys hy hulle eksperimenteel? Wat is die kliniese, filosofiese en biologiese implikasies daarvan?

SELYE SE VOORGANGERS

Selye erken dat hy nie oorspronklik is in die gedagte dat die liggaam beskik oor 'n aktiewe reaksievermoë teen skade nie. Meer as een het vroeër al daarna aandag getrek of probeer trek:

1. Hippokrates het, byvoorbeeld, daarvan gepraat dat daar gedurende siekte twee aparte elemente in die liggaam herken moet word: nl. *stryd,* sowel as *lyding.*

2. Claude Bernard het vir ons geleer van die liggaam se neiging om sy *milieu interieur* konstant te behou.

3. Cannon het dit in sy *Wisdom of the body* homeostase genoem.

4. John Hunter weer, het dit so gestel: ‚There is a circumstance in accidental injury which does not belong to disease — namely, that the injury done has in all cases a tendency to produce the disposition and the means of cure.'

Maar alhoewel dit niks nuuts is nie, is dit 'n waarheid, wat

ons met die meerdere wetenskaplike feitekennis as ons eerbiedwaardige voorgangers, dikwels in ons slimheid, minder duidelik bewus van is as wat hulle was, met hul minder kennis maar dieper wysheid.

Maar nie alleen lei Selye in sy genialiteit ons terug na die wysheid van Hippokrates nie — hy slaag ook daarin om dit met moderne wetenskaplike metodes te demonstreer en te verduidelik.

Voorgeskiedenis

Hoe kom Selye daartoe om hieroor navorsing te doen? Dit blyk dat dit die vrug is van sy ryk verbeelding, skerpsinnige insig, en onvermoeide praktiese ywer.

Daar was drie gebeurtenisse wat eintlik aanleiding gegee het tot sy idee.

Eerstens, as jong student in Praag het hy al gevoel dat daar 'n basiese onderliggende enersheid van net siekwees in alle pasiënte aanwesig is, behalwe hul verskillende simptome en tekens op die gronde waarvan hul siektetoestand gediagnoseer word — maar sy professore kon hom nie veel daaroor inlig nie.

Tweedens het hy dikwels gewonder oor die ongetwyfelde sukses deur die eeue heen van die non-spesifieke onwetenskaplike metodes van terapie wat aangewend word deur die toordokters en priesters en kwaksalwers, met hulle gebruik van toormiddels, non-spesifieke medikamente, bloedlating, en verhoging van liggaamstemperatuur. (Is ons huidige gebruik van insulien en elektriese skokbehandeling nie in elk geval soortgelyk nie?)

Die derde en kritieke fase in sy ontwikkeling, die ‚tide in the affairs of men, which taken at the flood leads on to fortune', kom egter in 1935 terwyl hy as jong medikus besig is met navorsing op die spoor van die ontdekking van 'n nuwe sekshormoen. Want, alhoewel sy poging om 'n nuwe sekshormoon te ontdek hopeloos faal, lei sy mislukking, soos in die geval van Fleming, met sy bedorwe stafilokkokke-kultuur, na 'n meer belangrike vonds.

In sy sekshormoon-eksperimente was hy besig om sy proefdiere met ekstrakte van ovariale weefsel in te spuit, toe hy 'n reeks van veranderinge oplet wat nog nie voorheen beskrywe is nie en wat by herhaling van die inspuiting elke keer konstant voorkom, nl.: (1) Vergroting van die byniere, (2) atrofie van die timusklier en ander limfoïde weefsel, en (3) die ontstaan van akute bloeding en ulserasie op die slymvlies van die ingewande.

Vereers was hy natuurlik in die wolke oor sy bevindinge, wat so konstant voorgekom het dat hy hul noodwendig as 'n gekoördineerde groep veranderinge moes beskou, d.w.s. as 'n triasformasie van 'n nuwe sindroom. As hy later die eksperimente herhaal met die inspuiting van plasentale weefsel, en weer dieselfde triasformasie opwek, steun dit sy vermoede, want hy het tog geweet dat plasentale sowel as ovariale weefsel in staat is om sekshormone af te skei.

Geïnteresseerd kyk hy vervolgens wat gebeur as hy hipofiseweefsel-ekstrak inspuit, en is bietjie verbaas om 'n herhaling van sy triasformasie te vind. Sou dit beteken dat die hipofise ook hierdie nuwe sekshormoon afskei? Maar dan ontwikkel sy steeds groeiende twyfel tot sekere onsekerheid as hy vind dat ook ekstrakte van milt, nier en enige ander orgaan-weefsel wat hy inspuit, dieselfde triasformasie aanbring. Verder let hy daarop dat hoe *suiwerder* die weefsel-ekstrak saamgestel is hoe *minder uitgesproke* die veranderinge, terwyl hoe meer *meer onsuiwer* die ekstrak hoe *meer uitgesproke* die veranderinge wat die triasformasie kenmerk.

Onder hierdie omstandighede begin hy maar te duidelik besef dat die waargenome triasformasie van veranderinge sekerlik nie die uitwerking van 'n nuwe sekshormoon kan voorstel nie, maar op die meeste die gevolge van een of ander gemeenskaplike weefselhormoon, en dan wonder hy verder: kon *dit* nie miskien die regte antwoord wees nie, want waarom was dit so opmerklik dat die onsuiwerste ekstrakte die triasformasie die duidelikste aangebring het?

As getroue navorser gaan hy onmiddellik voort met die verdere ondersoek van hierdie moontlikheid, en herhaal hy sy eksperiment nou met 'n inspuiting van verdunde formalien — 'n gifstof wat tog geen weefselhormoon kon bevat nie — en vind hy *nogeens* dat herhaalde inspuitings ook van formalien sy triasformasie van veranderinge aanbring: Bynier-vergroting, timo-limfatiese-involusie, en die vorming van hemorragiese ulserasie in die mukosa van die ingewande.

Die Geboorte van die Idee van Non-spesifieke ‚Stress'

Hiermee verdwyn Selye se drome van die ontdekking van 'n nuwe sekshormoon finaal.

Maar daarmee word ook gebore 'n *nuwe idee* — sou dit alles nie die antwoord wees op sy jare-ou-probleem van wat die basis is van ‚net-siek-wees' nie — die non-spesifieke ooreenkoms tussen verskillende siektetoestande, en die oplossing van die probleem waarom non-spesifieke terapie soms effektief kan wees? En opnuut gestimuleer, begin hy verdere eksperimente ontwerp en uitvoer om sy nuwe idee uit te toets: om te sien of daar wel so iets is as 'n basiese non-spesifieke groep van veranderinge in die liggaam wat veroorsaak word deur stimuli van uiteenlopende geaardhede.

EKSPERIMENTELE WAARNEMINGE

Proefdiere van verskillende soorte word nou onderwerp aan irritasie deur stimuli van allerlei aard, en die gevolge geduldig en noukeurig waargeneem, ge-analiseer en vergelyk, en die bevindinge gekorrelleer met mekaar, sowel as (sover moontlik) met dergelike waarnemings in die menslike liggaam onder soortgelyke omstandighede.

Aanvanklik is sy kollegas skerp krities oor sy onderneming — hulle beskou hom as nie minder as gek om 'n belowende loopbaan van hormoonnavorsing so prys te gee vir 'n nuttelose poging om, soos hulle dit beskrywe het: ‚to investigate the pharmacy of dirt'.

Hy volhard egter totdat hy uiteindelik erkenning ontvang. Nie alleen word bevestig dat die triasformasie van bynier-, timus-ingewandsveranderings konstant voorkom in die dier en menslike liggaam na irritasie met 'n wye reeks van stimuli nie (altyd natuurlik gesamentlik met die bykomstige spesifieke veranderinge eie aan die geaardheid van die betrokke stimulus wat aangewend is), maar oor die loop van jare slaag hy ook daarin om die verhouding en verwantskap van die waargenome veranderinge in fynere besonderhede te analiseer en hul kousale verband uit te pluis.

Die verskillende stimuli wat uitgetoets is, het o.a. die volgende ingesluit: Troumatiese skok; obstetriese skok; swaartegewig-skok; spier-oefeninge; aansteeklike siektes; bloedsverlies; senuwee-skok; blootstelling aan koue, sonstraal, X-strale, en radium-aktiwiteit; brandwonde; chemiese gifstowwe; bakteriële toksiene; tydelike afsnoering van bloedvate; vas en hongerly; verminderde suurstofspanning; en blootstelling aan vrees, humeursverlies, ens.

Voorwaar tog 'n uiteenlopende reeks van irriteermiddels, wat genoeg in geaardheid verskil om nie dieselfde gevolge te kan produseer nie. En tog, elke keer, afgesien van die verwagte kenmerkende spesifieke gevolge, kon Selye se non-spesifieke gekoördineerde veranderinge tot mindere of meerdere mate erken en gemeet en gedemonstreer word. Weliswaar het die veranderinge gewissel in intensiteit en duidelikheid, maar dit was net 'n verskil in graad van intensiteit — basies was hul almal dieselfde.

Die reaksie van die liggaam tot die irritasie van die daaglikse lewensprobleme is bv. minder uitgesproke as die non-spesifieke reaksies wat volg op blootstelling aan meganiese geweld, of 'n ernstige infektiewe siektetoestand, waar die gevolge ernstig genoeg mag wees om dodelik te wees. Maar basies kon aanwesigheid van dieselfde non-spesifieke veranderinge keer op keer herken word, en slaag Selye oortuigend daarin om die werklikheid te bewys van *stress* wat hy dan nou omskrywe as: ‚The common denominator of all adaptative reactions in the body; a state manifested by a specific syndrome which is made up by all the non-specifically induced changes within a biological system, after the variable specifically induced changes have been subtracted.'

Analise van Eksperimentele Bevindinge

Wanneer hy die besonderhede van die ‚non-spesifieke veranderinge' volledig analiseer, stel hy vas dat dit min of meer as volg ontwikkel: Dit is saamgestel uit sekere endokrien- en metaboliese veranderinge wat gekoördineerd die volgende ver-

loop volg: (Wysiginge en afwykings mag natuurlik voorkom, onder die invloed van inwendige sowel as uitwendige kondisionerende faktore, soos bv. oorerwing, vorige ondervinding, dieet, klimaat, sowel as die invloed soms van die bykomstige spesifieke veranderinge.)

Elke keer as die liggaam gestimuleer word, ondergaan dit beide spesifieke sowel as non-spesifieke veranderinge.

1. *Spesifieke gevolge* en reaksie wissel af volgens (*a*) die geaardheid van die stimulus en (*b*) die deel van die liggaam wat aangetas is. Bv. (*a*) trouma veroorsaak ander spesifieke veranderinge as 'n bakteriële indringer, en (*b*) veranderinge (spesifiek) as gevolg van irritasie van die longe sal verskil van die van die senuweegestel of die skelet en spiere.

2. Behalwe hierdie kenmerkende en wisselende spesifieke veranderinge, is daar altyd 'n bykomstige groep van *non-spesifieke* veranderinge — wat basies elke keer dieselfde is wat ook al die geaardheid van die stimulus, en hoe verskillend ook al die gedeelte van die anatomie wat direk aangetas word.

Hierdie *non-spesifieke* veranderinge, wat altyd dieselfde is, kom op hul beurt voor in *vier stadiums:*

(1) Die stadium van non-spesifieke *skade* of skok.

(2) Die stadium van *reaksie* teen en herstel van hierdie skade.

(3) Die fase van *verhoogde weerstand.*

(4) Die fase van *uitputting en oorgawe.*

Stadium 1 van Non-spesifieke Skade

Hierdie stadium word veroorsaak deur die tussenwerking van faktore waarvan die aard nog nie presies vasgestel is nie. Al wat bekend is, is dat hul van 'n non-spesifieke aard moet wees, en verder dat hul nie deur middel van die tussenwerking van òf die byniere òf die hipofise funksioneer nie, omdat hul plaasvind, na 'n stimulus, selfs in diere waarin die hipofise en byniere eksperimenteel vooraf uitgeskakel is.

Hierdie stadium van non-spesifieke skade sluit die volgende veranderinge in:

1. Die kliniese beeld van *skok,* met tagikardie, hipotermie, hipotensie, ens.

2. Verlies van proteïne en liggaamsgewig.

3. Die ontstaan van akute bloedings van en ulserasie in die slymvlies van die spysverteringskanaal.

4. Tydelike verhoging van plasma K.

5. Tydelike daling van plasma Cl.

6. Veranderinge in die tiroïed, pankreas en byniermedulla, ens.

Stadium 2 van die Reaksie en Herstel

Ook hier is dit nie bekend wat die geaardheid is van die primêre faktore wat hierdie stadium te voorskyn roep nie. Dit mag een of ander non-spesifieke metaboliet wees; dit mag selfs 'n faktor van 'n negatiewe soort wees, soos bv. 'n tekort aan sirkulerende kortikoïde. Ook is dit onbekend langs watter bane hierdie faktore hulle boodskappe gelei (waarskynlik *via* die bloedstroom). Ons weet egter wel die boodskap word nie oor die senuweebane gelei nie, omdat dié stadium eksperimenteel voorkom selfs na uitskakeling of onderbreking van die senuweebane.

Dit moet egter weer van 'n onspesifieke geaardheid wees omdat die gevolge eners is, wat ook al die aard van die oorspronklike irriterende stimulus.

Hierdie onbekende faktor, opgewek deur irritasie, stimuleer vervolgens die hipofise, want, as die hipofise uitgeskakel word, kom hierdie stadium nie voor nie. As gevolg van die stimulering van die hipofise, skei dié klier (1) *meer* kortikotrofiese hormone af en (2) word die vorming van ander hipofisehormone tydelik onderdruk. Daar is bv. verminderde afskeiding van (*a*) groeihormone — met inhibisie van groei, (*b*) gonadotrofiese hormone — met gevolglike amenoree, atrofiese testis, ens., en (*c*) laktogene hormone — met verminderde laktasie.

Uitwerking van Kortikotrofiese Hormone

Die toename in afskeiding van ACTH stimuleer die *bynierkorteks* se funksie. Die korteks raak vergroot, verloor vit. K en cholesterol, en skei meer kortikoïde-hormone af. Hierdie kortikoïde-hormone bestaan uit 'n mengsel van glukokortikoïde en mineraalkortikoïde. Hul is ook 'n mengsel van *anti-inflammatoriese* kortikoïde (A.K.) en *pro-inflammatoriese* kortikoïde (P.K.).

1. Deur die gluco-kortikoïde word die organiese CH_2-metabolisme gewysig (lewer en pankreas). Deur die mineraalkortikoïde, word die anorganiese en elektrolitiese metabolisme beïnvloed, en daardeur, sekondêr, die bloedvate, die hart, die niere, en die bloed.

2. Die P.K.s en A.K.s reguleer die inflammatoriese reaksies van die liggaam.

3. Die kortikoïde veroorsaak verder: Involusie en atrofie van die timus, limfoïde weefsel en, o.a. sirkulerende eosinofiele. D.w.s., die antiskok stadium bestaan uit: stimulus van hipofise — stimulus van korteks — kortikoïde afskeiding, metaboliese veranderinge + timo-limfatiese involusie.

Verdere Bespreking van Skok en Antiskok

Die skok-stadium se verskynsels is dus van 'n passiewe geaardheid, en stel voor tekens van non-spesifieke beskadiging. Dit mag enigiets duur van 'n paar minute tot oor die 24 uur. Ook mag dit *gering* van geaardheid wees — of so ernstig in graad dat dit *dodelik* is.

Wanneer dit egter nie dodelik is nie, word dit altyd opgevolg deur die stadium van *antiskok.*

Hierdie stadium bring verskynsels mee van 'n aktiewe deelname van die liggaam, en stel voor 'n reaksie teen die skade wat verrig is; as sodanig gaan dit gepaard met 'n terugkeer na normaal, of retrogressie van die verskynsels van skok, en wanneer geslaagd eindig dit met die *herstel van die non-spesifieke skade:*

Let wel: Hierdie twee stadiums van skok en antiskok is nie altyd duidelik van mekaar te onderskei nie, en die een of die ander mag om die beurt oorwegend voorkom. Maar, alhoewel dit so is dat hul gedeeltelik gemeng is en in mekaar inloop, is dit basies so dat die een op die ander volg, en dat die tweede eintlik die eerste neutraliseer en dat die tweede afhanklik is van die funksie van beide hipofise en bynier-korteks.

Uiteindelik, wanneer hierdie twee stadiums verby is, word gevind dat die liggaam intussen as gevolg daarvan nuwe status verwerf het, nl. die van *verhoogde weerstand.* Want, as die oorspronklike stimulus nou herhaal word, word gevind dat daar nie verder tekens van non-spesifieke skade of reaksie uitgelok word nie, en wel omdat gedurende hierdie verworwe toestand van verhoogde weerstand teen beskadiging, geen skade berokken word nie, en geen herstellende reaksie dus meer aangewakker word nie.

Met ander woorde, hierdie twee stadiums van skok en antiskok stel dus eintlik voor 'n gekoördineerde endokrien meganisme van die liggaam, waarmee hy sy verdedigingsmagte bymekaar roep, om dan volledig toegerus te wees en slaggereed te staan, ten volle weerstandig en geskik om verder enige toekomstige aanvalle af te weer sonder die opdoen van verdere skade — of reaksie tot skade.

Om dié rede noem Selye hierdie gekombineerde fase van skok en antiskok die fase van die *alarmreaksie,* die reaksie waarmee die noodseine uitgestuur word en die liggaam weerstandig en goed uitgerus nagelaat word in die daaropvolgende fase van *weerstand* (resistance).

Eksperimenteel egter duur hierdie fase van weerstand nie onbepaald voort nie. Want, as die irriterende stimuli voortdurend herhaal word, breek daar uiteindelik die tydstip aan wanneer die verworwe weerstand weer verdwyn en irritasie weer eens daarin slaag om tekens van skok en antiskok te verwek. Hierdie keer egter slaag die reaksie nie daarin om die skade te herstel nie en teen verdere skade te beskerm nie, en nou versink die liggaam in 'n finale fase van *uitputting,* oorgawe, en afsterwe (exhaustion).

Met hierdie verduideliking kan u miskien Selye se terminologie volg: *‚Stress* designates the sum of *all* the *non-specific* changes, effected by any factor which acts upon it. *A stressor* is any factor capable of acting upon the body and eliciting the changes of *stress.'*

‚G.A.S. The General Adaptation Syndrome is the sum of all the non-specific *systematic reactions* of the body, which ensue from long exposure to *stress.* This syndrome evolves in three stages:

‚1. *The alarm reaction* is the sum of all the non-specific phenomena elicited by *sudden* exposure to stimuli to which the organism is quantitatively and qualitatively not adapted. (*a*) Some of these phenomena are merely *passive* and as such represent signs of damage and shock; (*b*) others of these phenomena are *active* and as such represent signs of defence against shock — the counter-shock phase.

‚Either of these two phases may predominate; usually they are intermingled.

‚2. *The stage of resistance.* During this stage most of the morphological lesions of the alarm reaction regress and it is found that the organism's specific resistance, already starting during the defensive anti-shock stage, reaches its peak. This resistance cannot be maintained indefinitely, so if the stimulus continues to act, adaptation wears out and lesions characteristic of the shock phase, reappear.

‚3. *The stage of exhaustion* sets in with absence of resistance and finally death.'

PROBLEME EN IMPLIKASIES

Hiermee het ons dan kortliks die hooftrant van Selye se gedagtes probeer weergee. Met sy eksperimente het hy redelik oortuigend bewys gelewer van die aanwesigheid in die liggaam van 'n lewensbelangrike fisiologiese meganisme, en 'n bevredigende verklaring aangebied van hoe die meganisme gegrond is op 'n tussenwerking tussen die hipofise en bynier-korteks, en dat hierdie endokrien tussenwerking 'n essensiële rol speel in die liggaam se weerstandsvermoë teen skade beide gedurende siekte en gesondheid.

Daarmee het hy dan ook 'n belangrike bydrae gelewer tot ons insig in die geheime natuurkragte van ons wese, en voorsien hy ons met 'n hernude en versterkte vertroue in die natuur se vermoë om ons pasiënte te help herstel selfs in die gevalle waar ons bewus is van die onvoldoende hulp wat ons hul self kan aanbied.

Vervolgens, egter, kom ons tot die bespreking van sekere probleme i.v.m. die hele ‚stress'-meganisme wat nog nie voldoende opgelos is nie, en sekere kliniese implikasies wat Selye op sy bevindinge baseer — waaroor daar nog nie algemene ooreenstemming bereik is nie.

Om mee te begin is daar die raaisel van op welke manier hierdie non-spesifieke sindroom op enerse wyse deur so 'n verskeidenheid van stimuli aan die gang gesit kan word. Wat is die gemeenskaplike tussen-faktor? Selye stel die moontlikheid voor van die bestaan van 'n hipotetiese eenheid wat 'n onderdeel van die sel van die liggaamsweefsel vorm — hy noem dit 'n ‚reaktor', maar sy argumente daaroor is baie teoreties en ek persoonlik kon dit tot dusver nog nie begryp of aanvaar nie. Hy self erken dan ook dat dit tot so ver net 'n hipotese is, en dat hy nog soek na objektiewe eksperimentele bewys daarvoor.

Tweedens is daar Selye se verklaring van die ontstaan van die weerstandsfase wat volg op die alarmreaksie, in terme van 'n bron van aanpassingsenergie, wat beperk is in hoeveelheid, en wat deur die alarmreaksie beskikbaar gestel word. Dis ook op sterkte van hierdie bron van aanpassingsenergie wat hy sy ‚Way of life' uitwerk.

So skrywe hy bv.: ‚True age depends largely on the rate of wear and tear and on the speed of self-consumption; for life is essentially a process which gradually spends the given amount of adaptative energy which we inherited from our parents, which we name Vitality. This vitality is like a special kind of bank balance which you can use up by withdrawals, but cannot increase by deposits. Your only control over this most precious fortune is the rate at which you make your withdrawals. The solution certainly does not lie in stopping to withdraw, for this would mean death. Nor is it to withdraw just enough for survival, for this would permit only a vegetative life worse than death. The intelligent thing is to withdraw generously, but never to expend wastefully. Many people believe that after they have exposed themselves to very stressful activities, a rest can restore them to where they were before. This is false. Experiments on animals have clearly shown that each separate exposure leaves an indelible scar, in that it uses up reserves of adaptability which cannot be replaced.

‚It is true that immediately after some harassing experience rest can restore us almost to the original level of fitness by eliminating acute fatigue; but the emphasis is on the word 'almost'. Since we constantly go through periods of stress and rest in life, just a little deficit of adaptative energy adds up every day — it adds up to what we call ageing. What man must do is to equalize stress by deviation, and not allow any single part of his system to wear out before the rest. Vary your life, and don't allow one part to suffer from attrition.'

Dat dit alles interessant is en gesonde verstand aandui, gee ek geredelik toe, maar die argumente wat hy gebruik om die bestaan van sy ‚fund of adaptative energy' te bewys, sowel as sy stelling dat dit beperk in hoeveelheid is, vind ek nie orals logies nie. Daar is ongelukkig nie tyd om hierop verder in te gaan nie.

Ook sy verduideliking van hoe non-spesifieke terapie — beide die van die toordokters en van elektriese skok — werk, volg ek nie duidelik nie. Hy vergelyk die werking daarvan o.a. met 'n grammofoon wat nie werk nie, en dan weer begin speel nadat jy dit 'n stamp gegee het.

Laastens moet ons net 'n paar minute stilstaan by die kliniese implikasies van ‚stress'.

Selye stel voor dat as dit so is dat hierdie sindroom 'n lewensbelangrike funksie in die fisiologie van die liggaam vervul, dit goed denkbaar is dat enige afwykinge in die liggaam se vermoë om die meganisme normaal uit te voer, aanleiding mag gee tot abnormale of siektetoestande, net sowel as wat afwykinge van die funksie, bv. van die tiroïed òf tiroïtoksikose òf miksedeem sal meebring; en hierdie groep siektes sou hy dan wou klassifiseer onder die benaming: *Diseases of maladaptation.* (Let wel, nie ‚diseases of stress' nie — want stress is eerstens 'n fisiologiese toestand, en tweedens sou dit weer lei tot verwarring met toestande wat deur ‚mental stress' veroorsaak word.)

Onder hierdie klassifikasie kan 'n mens dan insluit:

1. *Simmonds* en *Addison* se siekte — omdat hul respektiewelik non-funksie van die hipofise en bynier voorstel met hul lae weerstandsvermoë teen enige stimulus.

2. *Waterhouse-Friderichsen* se sindroom van bloeding in die bynier sou moontlik kon beskou word as die gevolg van ooreising van die funksionerende bynier-korteks gedurende die ‚stress'-reaksie uitgelok met die stimulus van 'n kwaai meningitis of wat ook al, wat dan uitloop op die fase van uitputting.

3. Die akute *maag-ulkusse*, gepaard met brandwonde en bomaanvalle, sou inpas by die ingewandsulserasie beskrywe in die skade-fase van die alarmreaksie.

4. Dan raak hy nog meer gewaagd en stel voor dat, op die basis van sekere eksperimente, sekere gevalle van nefrosklerose, hipertensie, artritis, pankreatitis, eklampsie, en selfs uitsonderlike gevalle van tonsilitis en appendisitis, in terme van afwykings in die aanpassingssindroom sou kon verklaar word, maar erken dat hy nog maar gis, en nog nie volledige bewyse hiervoor kan lewer nie.

5. In die chirurgie is daar natuurlik die toepassing van kennis van die sindroom, waar ons vind dat pasiënte wie se byniere onder-normaal funksioneer, met kortisoon moet behandel word as ons nie wil hê dat hul met 'n minimale ingreep maksimale tekens van skok moet opdoen nie, bv. gevalle waar die bynier beskadig is deur organiese siektetoestande, of onderdruk is deur vroeëre kortisoon-behandeling.

Daar is ook voorstelle dat vetembolisme mag verwant wees aan bynieroreising gedurende die ‚stress' van trouma, en ons is bewus van die rol wat veranderinge in die eosinofiel-telling speel in die interpretasie van chirurgiese weerstandsvermoë.

Selye het ook interessante eksperimente uitgevoer om die verhouding van die kortikoïdehormoon tot die inflammatoriese

reaksie te bepaal, maar al hierdie interessante sake vereis 'n bespreking op hul eie.

SLOT

Met hierdie kort oorsig moet ek volstaan; my hoop is dat ek sommige van u genoeg geïnteresseer het om self verder oor ‚stress' te gaan nalees en nadink, en dan u eie oordeel te vorm oor die belang al dan nie van Selye se bydrae.

Op die voorblad van een van sy jongste uitgawes skryf Selye:

‚This book is dedicated to those:
who are not afraid to enjoy
the stress of a full life,
nor too naïve to think,
that they can do so
without intellectual effort.'

Mag ek, op my beurt, hierdie kort bespreking van Selye se werk opdra, as 'n vorm van hulde en klein blykie van waardering aan een van daardie soort mense na wie hy verwys, nl. dr. John v. d. Riet, die Ere-sekretaris van die Nagraadse Skool-beplanningskomitee, Bloemfontein.

STRANGULATED APPENDIX IN A HERNIAL SAC

I. G. SCHRAIBMAN, *formerly Registrar, Leicester General Hospital, Leicester, England*

The appendix is notoriously variable in its position, and its presence in a hernial sac, while uncommon, is within the personal experience of most surgeons.

Garland,[1] with transatlantic brevity, applied the term 'femoral appendicitis' to an inflamed appendix in a femoral hernia. This nomenclature could be extended as in inguinal appendicitis (indirect and direct), obturator appendicitis, and scrotal appendicitis, all of which have been described. The condition has unaccountably no eponymous association.

Of all cases of appendicitis, 0·13% occur in hernial sacs;[2] while the appendix is found in 0·77% of hernial sacs.[3] Some hernial appendices are uncomplicated; the rest may present with two complications, viz. acute appendicitis or strangulation of the appendix.

It is interesting to note that the first recorded appendicectomy was one in a hernial sac, performed by Claudius Amyand of St. George's Hospital, London, in 1735.[4] Many of the early appendicectomies were only incidental in that, being superficially situated in a hernial sac, they presented with suppuration or obvious swelling, and the exact diagnosis was made only at operation.

The following case is presented because it combines the features of an ectopic appendix in an indirect inguinal hernial sac, complicated by strangulation, in a neonate.

CASE REPORT

P.M.J., a 3-week-old male infant, was admitted to hospital on 20 May 1960 with a mass in the right groin for nine hours. The first symptom noted by the mother was repeated vomiting the night before admission. There was no constipation or passage of blood per anum.

The child was a full-term normal infant of 6½ lb. birth weight. Labour had lasted 62 hours and a pitocin drip was necessary. Since birth it had been breast fed.

On examination, the general condition was good; the abdomen was soft and not distended and the only positive finding was a tense mass in the position of a complete right indirect inguinal hernia.

Un er general anaesthesia ('fluothane', gas and oxygen *via* an endotracheal tube—Dr. F. Sambrook) the hernia was approached through an oblique inguinal incision. As soon as the external ring was opened, the contents of the hernia could be seen sliding back into the abdomen, so that the external ring must have been the constricting agent. The sac was opened forthwith and a quantity of yellowish, turbid, but not offensive, fluid escaped. The only contents were an oedematous, congested, plum-coloured appendix and the apex of the caecum which had undergone similar changes (Fig. 1).

The appendix was removed, but the stump was ligated and not inverted, because of the oedema of the caecal wall. The hernial sac was isolated and transfixed and the transversalis fascia was plicated with two No. 60 thread sutures medial to the internal ring. Apart from a mild wound infection, recovery was uneventful.

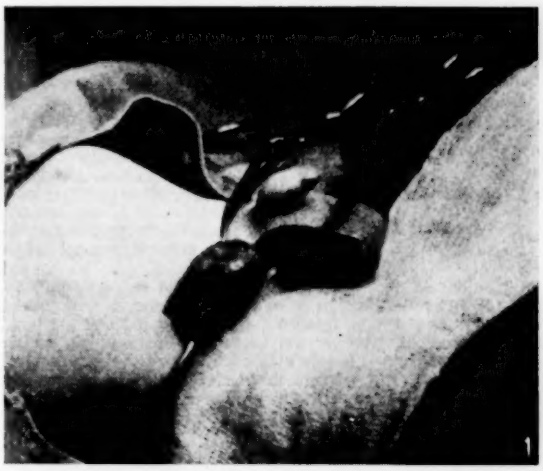

Fig. 1. Shows the darkened appendix and caecum presenting through the right inguinal incision.

DISCUSSION

The diagnosis can be made pre-operatively. As Reif[5] pointed out, if the primary complication is acute inflammation, abdominal symptoms will precede those localized to the site of the hernia, but when the hernial strangulation is primary, the abdominal signs will succeed the local ones. In a fair number, the diagnosis will not be made, but fortunately this is not of significance, for direct surgical attack is indicated in any case.

Reference to the literature reveals only 2 cases occurring at an earlier age than the one reported here. Reif[5] reported a gangrenous appendix in a scrotal hernia in a 2½-week-old child, and Beattie[6] described a strangulated appendix in an inguinal hernia in a premature infant of 3 weeks of age, 5 weeks before it was due to be born!

I should like to thank Mr. G. C. Sawyer for allowing me to treat and report this case.

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DIE EERSTE BEKLOPPING VAN DIE BORSKAS TWEEHONDERD JAAR GELEDE

J. G. STEYTLER, B.Sc. (STEL.), M.Sc., M.B., CH.B. (KAAPSTAD)

Departement van Mikrobiologie, Fakulteit van Geneeskunde, Universiteit van Stellenbosch en Karl Bremer-hospitaal, Bellville, Kp.

Leopold Auenbrugger (1722 - 1809), Oostenryker van geboorte, het in die jaar 1761 'n werk gepubliseer wat vir hom roem gebring het. Weliswaar het die idee van beklopping van die borskas eerste by hom ontstaan wat, as geneesheer van sy tyd, sonder enige hulpmiddels was in die diagnose en behandeling van longsiektes.

Dr. Auenbrugger

Gebore op 19 November 1722 in Graz, die seun van 'n hoteleienaar, gaan hy op jeugdige ouderdom na die Universiteit van Wenen, waar hy hom in sy mediese studies onderskei. Op 29-jarige ouderdom word hy internis-in-bevel van 'n hospitaal aldaar en tree 3 jaar later in die huwelik met 'n aantreklike, welgestelde dame. Lief vir die musiek en poësie, dog ook by uitstek 'n wetenskaplike, voer hy 'n kommervrye en gelukkige bestaan. As iemand van voortreflike karakter wat altyd bereidwillig was om sy medemens te help, word hy in 1784 in die adelstand opgeneem.

Met eindelose deeglikheid en nougesetheid in sy werk, toon hy veral belangstelling in afwykings van die borskas, wat, weens sy vaatjievormige fatsoen en in teenstelling met die buik, hom nie geredelik leen tot die betastende metode van ondersoek nie. Auenbrugger het gereeld die outopsies van sy pasiënte bygewoon. Instede van longe te sien wat gevul was met lug, was daar somtyds groot holtes teenwoordig, kolleksies van vog en etter, vergrotings van die hart, en aneurismas van die aorta; almal dinge wat gedurende die lewe *teenwoordig moes gewees het* maar waarvan hy as geneesheer onbewus was.

Gebaseer op sy proefondervindelike werk met die longe van kadavers, merk hy op dat wanneer die borskas van 'n gesonde persoon beklop word, dit 'n geluid voortbring soos ‚van 'n drom met 'n dik kleed oorgetrek'. 'n Dowwer klank of een van 'n hoër toonhoogte was vir hom, met sy fyn aanvoeling vir musikale klanke en skerpe waarnemingsin, 'n aanduiding van onderliggende patologie. Oor 'n tydperk van sewe jaar toets hy hierdie bevindinge deeglik uit, en beproef en ontleed hulle voordat hy hulle boekstaaf. In sy kort en saaklike *Inventum novum ex percussione thoracis* (1761) vind ons die eerste outentieke dokumentasie van die bekloppingstegniek as diagnostiese hulpmiddel met 'n uiteensetting daarvan soos ons dit vandag nog ken.

Misken deur sy tydgenote as iemand wat 'n diens aan die mediese wetenskap bewys het, is hy ongenaakbaar teenoor sy opposisie en openbaar hy 'n onwrikbare vertroue in homself en in sy eie opvattinge en doelstellinge. Eers in 1807 het sy metodes algemene inslag gevind en is die waarde van die bekloppingstegniek besef met die aanwending van diagnostiese en terapeutiese torakosintese as 'n direkte uitvloeisel hiervan. Kort voor sy dood in 1809 verskyn die Franse vertaling van sy boek deur Corvisart en word Auenbrugger daardeur een van die weiniges van sy tyd wat erkenning van sy werk belewe.

In hierdie jaar word die verskyning van sy nederige manuskrip dus herdenk, wat, alhoewel eenvoudig en elementêr op die oog af, tog as 'n pionierswerk beskou moet word in die geneeskunde.

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PASSING EVENTS : IN DIE VERBYGAAN

Dr. D. P. Knobel, seun van dr. W. F. Knobel van Pretoria, het so pas sy finale eksamen in die geneeskunde in Edinburg met sukses afgelê. Op die oomblik kuier hy met gade en seuntjie by sy ouers voordat hy terugkeer om sy hospitaaldiensjaar daar te voltooi.

* * *

University of Cape Town and Association of Surgeons of South Africa (M.A.S.A.), Joint Lectures. The next lecture in this series will be held on Wednesday 13 September at 5.30 p.m. in the E-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape. Mr. W. M. Schulze will speak on 'Conservative management of peripheral vascular disease'. All members of the Medical Association are welcome to attend this lecture.

* * *

Mr. Felix Machanik, orthopaedic surgeon, of Johannesburg and Springs, has returned to South Africa after a three months' study tour of Great Britain and Europe, where he visited many orthopaedic centres, clinics and hospitals.

* * *

The Southern African Cardiac Society. At a meeting of the Western Province Section of this Society, held on Thursday 17 August at Groote Schuur Hospital, Dr. Leon Isaacson gave a talk on 'Vectorcardiography'. The lecture was illustrated by an analysis of about 50 cases of ischaemic and congenital heart disease. The traces were photographed on a cathode-ray oscilloscope.

* * *

Mr. Michael Katzen, surgeon, of Johannesburg, wishes to advise his colleagues that his telephone numbers have inadvertently been omitted from the latest telephone directory. They are: Rooms 220428, residence 421149, emergency 224191.

Dr. A. Schiller, ear, nose and throat surgeon, of Cape Town, has returned from a short overseas visit and has resumed his practice.

* * *

National General Practitioners Group (M.A.S.A.). The Annual General Meeting of this Group will be held on Monday 25 September 1961 at 2.15 p.m. in Room F, Arts Block, University of Cape Town, Rondebosch, Cape.

* * *

Mr. D. R. Barnes, urologist, of Cape Town, has changed his address to 419 Medical Centre, Heerengracht. His telephone numbers remain unchanged.

* * *

South African Medical and Dental Council. A vacancy has arisen among the elected medical practitioners on the Council, owing to the resignation of Dr. James Black. Every medical practitioner resident in the Republic of South Africa is eligible for nomination, and the closing date for nominations is 23 September 1961. A notice in the *Government Gazette* of 1 September 1961 gives details of how the nominations should be effected.

Suid-Afrikaanse Mediese en Tandheelkundige Raad. As gevolg van die bedanking van dr. James Black het 'n vakature ontstaan in die gekose geneeshere op die Raad. Alle geneeshere wat in die Republiek van Suid-Afrika woonagtig is, kan genomineer word vir verkiesing, en die sluitingsdatum vir nominasies is 23 September 1961. Besonderhede oor die prosedure insake die nominasies verskyn in 'n kennisgewing in die *Staatskoerant* van 1 September 1961.

The Seventh International Congress for Internal Medicine will take place under the Chairmanship of Professor Dr. Wollheim, Würzburg, from 5 to 8 September 1962 in Munich (Federal Republic of West Germany).

The main topics to be discussed are: (*1*) Immunity processes in the pathogenesis of internal diseases, (blood diseases, kidney diseases, thyroid diseases, heart diseases, vascular diseases); (*2*) shock and collapse in internal medicine (pathogenesis, haemodynamics, clinical forms and consequences of shock and their therapy); and (*3*) environment and disease (rheumatism, hypertension, diabetes, arteriosclerosis, infarction of the myocardium).

Further information is available from the Secretariat of the Seventh International Congress for Internal Medicine, Schwalbacherstrasse 62, Wiesbaden, Deutsche Bundesrepublik; Telegraphic address: INTERMED, Wiesbaden.

The South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting. The next meeting will be held on Monday 18 September 1961 at 5.10 p.m. in the Institute Lecture Theatre. Dr. Mary Salkinder will speak on 'Indications of a viral aetiology in some cancers'.

* * *

Research Forum, University of Cape Town. The next meeting will be held on Thursday 14 September 1961 at 4 p.m. in the Tutorial Room of the Pathology Department, Medical School, Observatory, Cape. Dr. E. Dowdle will speak on 'The metabolism of glycine in experimental porphyria'. This will be followed by a Staff Clinical Conference at 5.10 p.m. in the Falconer (E-floor) Lecture Theatre, Groote Schuur Hospital, Observatory, Cape. All who are interested are invited to attend these meetings.

UNIVERSITY NEWS : UNIVERSITEITSNUUS

UNIVERSITY OF CAPE TOWN MEDICAL HISTORY CLUB

On Wednesday night, 16 August, 30 enthusiastic persons met informally in the Doctors' Room of the Medical Library in Observatory and discussed the formation, scope, and organization of a Medical History Club. In general it was agreed to base the Club on the Medical School at the University of Cape Town where the Medical Library, with its admirable medical historical collection and its comfortably furnished Doctors' Room, would be a logical meeting place. Membership would be open to all interested persons and not only to members of the University of Cape Town or to members of the medical profession; in fact, the participation of doctors' wives, of medical students, and of lay persons was particularly to be encouraged. It was decided that the Club would arrange small seminars for the intimate study of specialized topics as well as general meetings where lectures on more popular subjects would be given.

A steering committee was elected to arrange the first few meetings, pending the election of a more constitutional executive. The steering committee consists of Dr. Hymie Gordon (convener), Miss Sheila Katcher (secretary), and Dr. A. P. Blignault. Interested persons are invited to communicate with the secretary at the Medical Library, Anzio Road, Observatory, Cape (telephone 55-4929), or with either of the other members of the steering committee.

SISTERS INCORPORATED

UNMARRIED MOTHERHOOD

On Thursday 28 September 1961 an interesting symposium will be held in the large lecture theatre in the Arts Block at the University of Cape Town. The Organizing Committee of Congress has invited an organization known as 'Sisters Incorporated' to be responsible for this symposium, and the programme will be found below.

'Sisters Incorporated' is an organization of doctors' wives and others who have undertaken to care for unmarried mothers in Cape Town. They are hoping that doctors attending Congress will be interested in their work and that the wives of doctors in other centres may be encouraged to undertake similar work in their areas.

The symposium will commence at 9.15 a.m.

PROGRAMME

The full programme is as follows:

1. Opening remarks by the Chairman, Dr. A. H. Tonkin, Secretary of the Medical Association of South Africa.
2. 'The needs of the unmarried mother', by Dr. Owen le Roux, Ph.D., of the Christelike Maatskaplike Raad.
3. 'Medical aspects of pregnancy in the unmarried mother', by Dr. H. J. H. Claassens, M.Med. (O. & G.), M.R.C.O.G., F.C.O. & G. (S.A.).
4. 'The legal aspects of adoption', by Mr. J. Horne, Commissioner of Child Welfare.
5. 'Adoption procedure by an approved Child Welfare agency and the problems of private placement', by Dr. Adele Impey, Chairman of the National Council for Child Welfare Standing Committee on Adoptions.
6. 'Sisters Incorporated — a registered welfare organization to assist the unmarried mother', by Mrs. S. Henderson, the Chairman of Sisters Incorporated.

There will be an interval during which tea will be served, and time for questions and answers will follow.

Further information in the work of Sisters Incorporated may be obtained at their stall at the Scientific Exhibition in the lower Zoology Laboratory.

DIE ONGEHUDE MOEDER

Op Donderdag 28 September 1961 sal 'n interessante simposium gehou word in die Groot-lesingsaal in die Gebou vir Kunste by die Universiteit van Kaapstad. Die Organiserende Komitee van die Kongres het 'n organisasie bekend as ‚Sisters Incorporated' uitgenooi om hierdie simposium te organiseer, en die program word hieronder geplaas.

‚Sisters Incorporated' is 'n organisasie van die vrouens van dokters en andere wat onderneem het om te sorg vir ongehude moeders in Kaapstad. Die organisasie hoop dat dokters wat die Kongres bywoon in die werk van die organisasie sal belang stel en dat die vrouens van dokters in ander sentrums aangemoedig mag word om soortgelyke werk te onderneem in hul gebiede.

Die simposium begin om 9.15 vm.

PROGRAM

Die volle program is soos volg:

1. Opening van die simposium deur die Voorsitter, dr. A. H. Tonkin, Sekretaris van die Mediese Vereniging van Suid-Afrika.
2. ‚Die behoeftes van die ongehude moeder', deur dr. Owen le Roux, Ph.D., van die Christelike Maatskaplike Raad.
3. ‚Mediese aspekte van swangerskap by die ongehude moeder', deur dr. H. J. H. Claassens, M.Med. (O. & G.), M.R.C.O.G., L.K.O. & G. (S.A.).
4. ‚Regsaspekte van aanneming', deur mnr. J. Horne, Kommissaris van Kinderwelvaart.
5. ‚Die prosedure van aanneming deur 'n goedgekeurde Kinderwelvaartvereniging en die probleme van private uitplasing', deur dr. Adele Impey, Voorsitter van die Permanente Komitee vir Aanneming van die Nasionale Raad vir Kinderwelvaart.
6. ‚Sisters Incorporated — 'n geregistreerde welvaartorganisasie wat die ongehude moeder help', deur mev. S. Henderson, Voorsitter van ‚Sisters Incorporated'.

Daar sal 'n teepouse wees, en tyd vir vrae en antwoorde sal dan volg. Verdere informasie in verband met die werk van ‚Sisters Incorporated' kan verkry word by hulle stalletjie by die Wetenskaplike Uitstalling in die onderste Soölogie-laboratorium.

IN MEMORIAM

DR. SAM GEFFEN, M.B., CH.B. (DUBLIN), D.A. (R.C.P. & S.) (LOND.)

Dr. J. Abelsohn, of Cape Town, writes:

The death occurred recently at Groote Schuur Hospital of Dr. Sam Geffen, formerly of Johannesburg and Durban.

Born in Paarl 65 years ago, Dr. Geffen commenced his studies at the University of Cape Town and then proceeded to Dublin, graduating in 1921. He practised in Paarl for two years, subsequently settling in Johannesburg, where he built up an extensive practice.

Dr. Geffen

In 1938, at the age of 42, he decided to specialize in anaesthetics, and, after proceeding to London, where he spent a year doing postgraduate study, he obtained the Diploma of Anaesthetics of the Royal Colleges of Surgeons and Physicians. At that time the D.A. (R.C.P. & S.) had only very recently been introduced — Dr. H. Grant-Whyte of Durban being the first South African to obtain this diploma by examination in 1936, I followed in 1938, and Dr. Geffen in 1939.

During the years 1939 — 1951, by dint of hard conscientious work, he built up a wide circle of friends and a very large anaesthetic practice in Johannesburg. He contributed very greatly at that time by introducing new ideas and new techniques — the use of 'pentothal', not only for induction but also for maintenance of anaesthesia; blind intubation; the Etherington-Wilson technique for high spinals; and the administration of avertin and cyclopropane.

I first met Sam Geffen doing the rounds of the London Hospitals in 1938, and over the years a staunch and sincere friendship grew between us. Devoted to his family and his work, he was gifted with high qualities of integrity and sincerity, being imbued with a shrewd perception of the human problems and anxieties of his patients, about to undergo the ordeal of major surgery. By his simplicity and sincerity he was able to inspire confidence and allay their fears.

From 1940 - 1950 he was associated with Dr. Lee McGregor, who had become the recognized South African authority on thoracic sympathectomy for the relief of hypertension.

The stress and strain of his work unfortunately exacted its heavy toll, and in 1951, following a severe attack of coronary thrombosis, he was left with a failing heart and was never able to resume his practice.

He faced each setback philosophically, and his unfailing sense of humour helped to sustain him during the many years of his illness, which imposed upon him a very restricted life; at periods he actually spent more time in than out of his bed.

Dr. Geffen leaves his wife and a married daughter, to whom we express our heartfelt sympathies and in whose sorrow we share.

'Let us not grieve that he is dead — but rather rejoice that he had lived'.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

ADROYD

Parke, Davis have introduced Adroyd, a newly developed steroid with potent tissue-building properties, and supply the following information:

Description: Adroyd (oxymetholone) is a partially synthetic steroid (17β hydroxy-2-hydroxymethylene-17α-methylandrostan-3-one) which exhibits a powerful protein anabolic tissue-building effect.

Adroyd is designed primarily for the individual who requires supportive treatment to help restore and maintain nitrogen equilibrium. Its action is characterized by the formation of new tissue (chiefly muscle), by weight gain, and by a decrease in the urinary excretion of nitrogen, potassium, phosphorus and calcium.

Unlike most anabolic agents having an androgenic activity, Adroyd has a high degree of anabolic activity but a low degree of masculinizing properties. In both experimental studies and clinical trials it consistently demonstrated a high degree of anabolic activity coupled with a low order of androgenicity, when administered in recommended doses.

Indications. Adroyd is useful in a diverse group of clinical conditions where an unfavourable or negative nitrogen balance is manifested. Among such conditions are asthenia, carcinomatosis, chronic diseases such as tuberculosis, sprue, Still's disease, the catabolic phase during the recovery period following surgery, recovery from severe infectious diseases, recovery from severe burns, fractures, and osteoporosis. It is useful pre-operatively, especially in patients who have lost tissue from the disease process itself or from associated symptoms such as anorexia. It may stimulate appetite and weight gain in underweight individuals and it has been administered either alone or in combination with supplementary vitamin and mineral therapy.

Dosage and administration. Dosage varies from 2·5 mg. to 10 mg. per day, administered orally either before or with meals. No untoward gastro-intestinal side-effects have been recorded.

Precautions. Because Adroyd retains some — though small — androgenicity, it shares with all androgens the tendency to salt retention. It should, therefore, be used with caution in persons with cardiac disease. Caution should also be observed in cases of nephritis and nephrosis.

Because of changes which have been observed in hepatic function on long-term use with Adroyd at higher doses, care should be observed in patients with known hepatic damage. Since very young and pre-adolescent individuals are unusually sensitive to the masculinizing effects of androgens, they should be under careful supervision during therapy.

As with all substances having androgenic effect, Adroyd is contraindicated in patients with prostatic carcinoma.

Presentation. Adroyd is available in 5 mg. scored tablets, in bottles of 15 and 100.

Further information may be obtained from Parke, Davis Laboratories (Pty.) Ltd., P.O. Box 24, Isando, Transvaal.

BOOK REVIEWS : BOEKBESPREKINGS

BEHANDELING

The Modern Treatment Yearbook 1961. Ed. by Sir Cecil Wakeley, Bt., *K.B.E.*, C.B., LL.D., M.Ch., D.Sc., F.R.C.S., F.R.S.E., F.R.S.A., F.A.C.S. and F.R.A.C.S. Pp. ix + 310. Illustrated. R3.50 net. London: Baillière, Tindall and Cox Ltd. 1961.

Hierdie jaarlikse publikasie handhaaf sy standaard nogeens, om 'n breë oorsig te gee wat baie afdelings van medisyne dek. Die verskillende artikels gee 'n goeie aanduiding van die nuutste behandeling, maar dit tref 'n mens dat die ou paaie nog in baie opsigte as die beste geld, b.v. die oorsig in verband met aambeie. Dit tref 'n mens dat die boek, uiteraard, geskryf is vir toestande in Engeland, waar, onder die Nasionale Gesondheidsdienste, die algemene praktisyn soms degenereer het tot 'n sorteer-beampte wat die pasiënte net na die regte spesialiste moet verwys, deur wie die eintlike behandeling dan

onderneem word. Die boek bied vir hulle 'n goeie aanduiding wat gedoen kan word en lê die nodige klem op die ondersoekmetodes en die differensiële diagnoses.

Die hoofstuk oor stafilokokkale infeksie van die vel stel teleur, want net 'n paar antibiotikas word gemeld, en die waarde van 'n outogene vaksine word nie hoog geskat nie. Niks word gemeld van die kulture vir sensitiwiteitstoetse van die bakterieë nie, en laasgenoemde is dikwels nodig om 'n kwaadaardige stafilokokkale infeksie van die vel behoorlik te behandel. I.S.S.

CHEMISTRY OF THE NORMAL AND THE FAILING HEART

Chemistry of Heart Failure. By William C. Holland, M.D., and Richard L. Klein, Ph.D. Pp. xiii + 116. 16 illustrations. R4.40. Springfield: Charles C. Thomas. Oxford: Blackwell Scientific Publications. 1961.

In the foreseeable future the detailed chemistry of most biological processes, including muscle contraction, may be elucidated. But with all the chemistry known there will still be problems as to how the processes are effected and how the cellular organization is maintained.

In this monograph the authors carry the reader from the macroscopic through the microscopic, then to the molecular and submolecular level, in their attempt to explain the processes associated with contraction of the normal and the failing heart. They have introduced the concept of free energy.

A knowledge of physics and chemistry is required for the understanding of basic thermodynamic and chemical considerations of the normal and failing heart, and the latest information, supported by many references to the literature, is presented in this book. The mode of action of digitalis and the ionic and metabolic events accompanying fibrillation are also discussed. Clinicians, cardiologists, chemists, and physicists will find valuable information in this concise monograph. N.S.

SURGERY OF THE ANUS, RECTUM AND COLON

Surgery of the Anus, Rectum and Colon. By J. C. Goligher, Ch.M., F.R.C.S. Illustrated. Pp. vii + 829. R16.80. London: Cassell. 1961.

In this well-produced and comprehensive book the anatomy is first reviewed in detail and there follow some 10 chapters on the minor ano-rectal conditions. While there must obviously be minor points of difference between surgeons here, nothing must detract from the wealth of detail and practical information so lucidly presented in this section.

In the chapters on carcinoma of the colon and rectum, Duke's superb work at St. Marks Hospital is extensively quoted, and all the problems from early diagnosis to final treatment are fully covered. Virtues of the valuable extended left hemi-colectomy and details of the technique are clearly set out. The treatment of carcinoma of the rectum in terms of sphincter-saving operations, in which Professor Goligher has extensive experience, is covered in great detail, and all the standard operations for carcinoma of the rectum are very adequately described and illustrated.

The surgical treatment of ulcerative colitis is well presented except that there is little discussion on restorative operations with avoidance of ileostomy. There is also a standard chapter on diverticulitis, stressing the case for earlier and more frequent one-stage surgery. Few surgeons in training and few young consultants, for whom this excellent book has especially been written, can afford to be without it. A.B.

BOOKS RECEIVED : BOEKE ONTVANG

A Text-book of Clinical Pathology. 6th edition. Edited by Seward E. Miller, M.D. Pp. xxi+894. Illustrated. R12.00. London: Baillière, Tindall and Cox Ltd. 1961.

Bulletin of the Medical Library Association. The National Library of Medicine—Index Mechanization Project. Pp. 96. Illustrated. Washington: The National Library of Medicine. 1961.

Medical Entomology. 5th edition. By William B. Herms, Sc.D. Revised by Maurice T. James, Ph.D. Pp. xi+616. Illustrated. New York: MacMillan. 1961.

Whillis's Elementary Anatomy and Physiology. 5th edition. By Roger Warwick, B.Sc., Ph.D., M.D. vii+275. R2.40. London: J. & A. Churchill. 1961.

Haemophilic Diseases in Denmark. By Knud-Erik Sjolin, M.D. Pp. 349. Illustrated. R3.50. Oxford: Blackwell Scientific Publications. 1960.

Progress in Clinical Surgery. Series II. Edited by Rodney Smith, M.S., F.R.C.S. Pp. x+327. Illustrated. R5.00. London: J. & A. Churchill Ltd. 1961.

Progress in Clinical Medicine. 4th edition. By various authors. Ed. by Raymond Daley, M.A., M.D. Cantab., F.R.C.P., and Henry Miller, M.D. Durh., F.R.C.P., D.P.M. Pp. x + 345. 25 illustrations. R5.00 net. London: J. & A. Churchill 1961.

Symptom Diagnosis. 5th edition. By Wallace Mason Yater, A.B., M.D., M.S. (in Med.), F.A.C.P., and William Francis Oliver, B.S., M.D., F.A.C.P. Pp. x + 1035. $15.00 net. New York: Appleton-Century-Crofts. 1961.

The British Encyclopaedia of Medical Practice. 2nd edition. Pharmacopeia 1961. Pp. viii + 1006. Durban: Butterworths and Co. 1961.

The Public Health Inspector's Handbook. 10th edition. A Manual for Public Health Officers. By Henry H. Clay, F.R.S.H., F.I.P.H.E. Pp. xvi + 673. 101 illustrations. R5.50. net. London: H. K. Lewis. 1961.

Fearon's Introduction to Biochemistry. 4th edition. By William John Edward Jessop, M.Sc., M.D., D.Ph. Pp. vi + 470. R3.00 net. London: William Heinemann. 1961.

Essentials of Materia Medica, Pharmacology and Therapeutics. 8th edition. By R. H. Micks, M.D., F.R.C.P.I. Pp. xii + 444. R3.00 net. London: J. & A. Churchill. 1961.

Clinical Endocrinology. 3rd edition. By Laurence Martin, M.D. (Camb.), F.R.C.P. (Lond.). Pp. vii + 275. 48 illustrations. R2.80 net. London: J. & A. Churchill. 1961.

Common Diseases of the Ear, Nose and Throat. 3rd edition. By Philip Reading, M.S. (Lond.), F.R.C.S. (Eng.). Pp. viii + 264. Illustrated. R2.40 net. London: J. & A. Churchill. 1961.

Direct Analysis and Schizophrenia. Clinical Observations and Evaluations. By O. Spurgeon English, M.D., Warren W. Hampe, Jr., M.D., Catherine L. Bacon, M.D., and Calvin F. Settlage, M.D. Pp. vii + 128. $4.25. London and New York: Grune and Stratton. 1961.

Le Kyste Hydatique. Thérapeutique Chirurgicale. Par. P. Goinard, J. Pegullo, G. Pélissier. Un Volume de 204 pages, avec 90 figures. Paris: Masson et Cie. 1961.

Miscellaneous Notes. (Eleventh Series). By F. Parkes Weber, M.D., F.R.C.P., F.S.A. Pp. 8. 25c. London: H. K. Lewis. 1961.

Pharmacology for Nurses. 2nd edition. By J. R. Trounce, M.D., M.R.C.P., Pp. viii + 296. Illustrated. R1.60. London: J. & A. Churchill. 1961.

Baillière's Pocket Book of Ward Information. 10th edition. Revised by Marjorie Houghton, M.B.E., S.R.N., S.C.M., D.N. (Lond.) Pp. 221. 65c. Postage 7½c. London: Baillière, Tindall and Cox. 1961.

A Manual of Pharmacy Law. 2nd edition. By T. W. Price, M.A., LL.B., Ph.D. (Cantab.), B.A., LL.B., LL.D. (South Africa), and R. Pannall, Dip. Pharm. (S.A.); M.P.S. Pp. xxii + 249. Durban: Butterworths. 1961.

Modern Surgery for Nurses. 5th edition. Ed. by F. Wilson-Harlow, M.B., B.S. (Dunelm), F.R.C.S. (Eng.). Pp. xxiii + 883. Illustrated. R3.00 net. London: William Heinemann. 1961.

The Scientific Basis of Medicine Annual Reviews 1961. British Postgraduate Medical Federation. Pp. xi + 342. Illustrated. R4.00. London: The Athlone Press. 1961.

Vertebrate Biochemistry. In preparation for medicine. By M. W. Neil, Ph.D., F.R.I.C. Pp. xii + 360. Illustrated. R4.00 net. London: Pitman Medical Publishing Co. 1961.

A Guide to Cardiology. By J. C. Leonard, M.D. (London), M.R.C.P. (London), and E. G. Galea, M.B. (Queensland), M.R.C.P. (London), M.R.A.C.P. Pp. xii + 267. Illustrated. R2.75 net. Edinburgh and London: E. & S. Livingstone. 1961.

Systematic Observation of Gross Human Behavior. By G. R. Pascal and W. O. Jenkins, Professors of Psychology, University of Tennessee. Pp. ix + 126. $4.75. London and New York: Grune and Stratton. 1961.

Physiology of the Salivary Glands. By A. S. V. Burgen, M.D. (Lond.), M.R.C.P. and N. G. Emmelin, M.D. (Lond.). Pp. 279. Illustrated. R3.50 net. London: Edward Arnold Publishers. 1961.

Optics. An introduction for ophthalmologists. By Kenneth N. Ogle, Ph.D. Pp. xv + 265. Illustrated. R7.00. Oxford: Blackwell Scientific Publications. 1961.

The Physics of Radiology. 2nd edition. By Harold Elford Johns, M.A., Ph.D., F.R.S.C., LL.D. Pp. xvi + 767. Illustrated. R18.40. Oxford: Blackwell Scientific Publications. 1961.

Kernicterus and its Importance in Cerebral Palsy. A Conference Presented by The American Academy for Cerebral Palsy. 11th Annual Meeting, New Orleans, Louisiana. Pp. xi + 306. Illustrated. R7.00 net. Oxford: Blackwell Scientific Publications. 1961.

Chemistry of Enzymes in Cancer. By Franz Bergel, D. Phil. Nat., D.Sc., F.R.S. Pp. xi + 122. Illustrated. R4.40 net. Oxford: Blackwell Scientific Publications. 1961.

The Chemistry of Brain Metabolism in Health and Disease. By J. H. Quastel, Ph.D., D.Sc., F.R.S.C., F.R.S. and David M. J. Quastel, M.D., C.M. Pp. xi + 170. R5.20. Oxford: Blackwell Scientific Publications. 1961.

Regional Block. A Handbook for Use in the Clinical Practice of Medicine and Surgery. 3rd edition. By Daniel C. Moore, M.D. Pp. xvi + 393. Illustrated. R10.00. Oxford: Blackwell Scientific Publications. 1961.

Personality, and Success in Marriage. By Robert E. Morton. Pp. 110. R1.00. London: William Heinemann. 1961.

CORRESPONDENCE : BRIEWERUBRIEK

TESTING SCHOOL CHILDREN'S VISUAL ACUITY

To the Editor: Each year I see a number of children whose parents have been notified by the school that they have some visual defect. Unfortunately among this number there are a few in whom the defect has been discovered too late to remedy.

As is well known, the child from birth to about the age of 6 years is developing the use of the eyes and if, for any reason, one eye is not used as much as the other, as might occur in cases of squint or marked difference in refraction between the eyes, the neglected eye remains functionally undeveloped — the so-called amblyopia ex anopsia. The main treatment for this condition is occlusion of the more developed eye, and the degree of success is inversely proportional to the age of the child when treatment is begun. In the child over 6 years the prognosis is very poor and worsens rapidly.

With the purpose of finding these cases sooner, the Bureau for the Prevention of Blindness has been instrumental in introducing into the curriculum of training of nursery-school teachers the testing of visual acuity. Thus the nursery-school teachers themselves will be able to test their charges. It is hoped that this will become the recognized practice throughout South Africa. Unfortunately, however, the nursery schools at present cater only for a small minority of preschool children. However, as most 5-year-olds are nowadays admitted to primary schools, there can be no reason why these teachers, too, should not be trained to test visual acuity. As it is, part of their training is the elementary anatomy and physiology of the eyes. The principles and methods of testing the visual acuity would not demand too much extra time and the benefits would be of great value. At present the school medical officers and their personnel do these tests. It is obvious that they cannot cater for all schools within a reasonable period, and up till now children have been 9 and even 10 years old before being tested for the first time. Apart from testing the 5- and 6-year-olds, every child should be tested each year. Myopia and other defects often develop during these years. A child's progress might be hampered for several years before the fault is discovered.

The question of the standards set for the referral of such cases of visual defect to the ophthalmologist need not be discussed here. It is obviously a matter that must be considered very carefully, since no parent relishes the expense of an unnecessary medical investigation.

The main intention of this report is to draw the attention of the medical profession and the public to this need and how it could be solved. That lay people should test the visual acuity is nothing new. It is done daily by opticians who have trained themselves to do this efficiently. Industry would be well advised to have suitable tests as a condition of employment. Each year motor-vehicle drivers sign a statement that their vision is not impaired. Unfortunately, only too often an individual is not aware of the deterioration or its degree. A few years ago I saw an adult who was hypermetropic and without glasses could see only 6/60. With spectacles he saw 6/6. About 4 years previously he had obtained glasses from an optician for reading only. At that time he saw 6/9 without glasses. This was adequate vision for him to drive a car at that time, but when I saw him his acuity of 6/60 was certainly insufficient, yet he was unaware that he was unfit to drive. One can only surmise that, had he been wearing glasses constantly, an accident, in which he had been blamelessly involved, might have been avoided had he had just that keener sight, and 2 lives might have been saved.

The pace of modern life demands good sight. There is no reason why each and every school should not be able to test its own pupils. There is every reason for the various Provincial authorities to demand that all school teachers be trained to do such tests.

E. Epstein

Medical Centre
Jeppe Street
Johannesburg
25 August 1961

DIFFERENTIAL SALARY SCALE

To the Editor: Your correspondents, E. B. Dowdle and E. Barrow in their letters[1] on differential salary scales for White and non-White medical officers at New Somerset Hospital (in the *Journal* of 19 August 1961) may have given the impression that the instance they cite is exceptional. It is perhaps not generally known that differential salary scales are used in both the state and the provincial medical services in this country. In the two services the principle of differentiation has been refined so as to distinguish not only between 'White' and 'non-White' medical officers, but also between 'Coloured/Asiatic' and 'Bantu'.

It is shameful that such discrimination should exist, and it is to be hoped that the medical profession in South Africa will succeed in their efforts to ensure that salaries are allotted solely on the basis of merit.

P. B. Neame

22 Abelia Road
Kloof, Natal
28 August 1961

1. Correspondence (1961): S. Afr. Med. J., **35**, 700.